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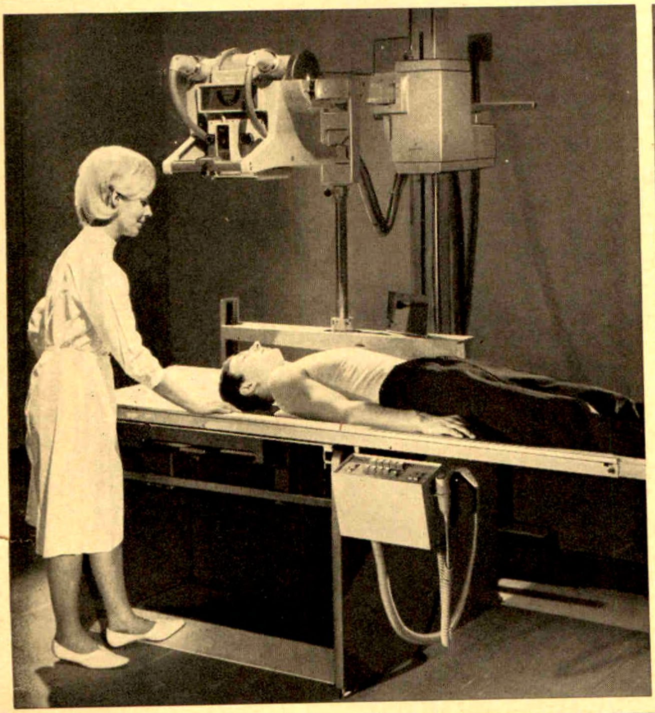

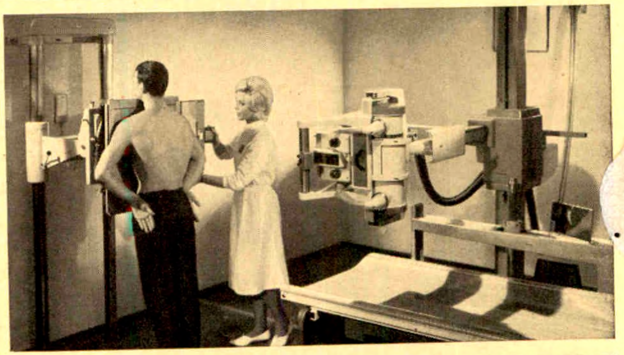

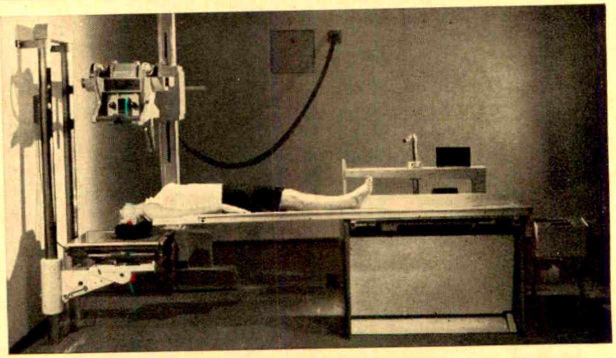

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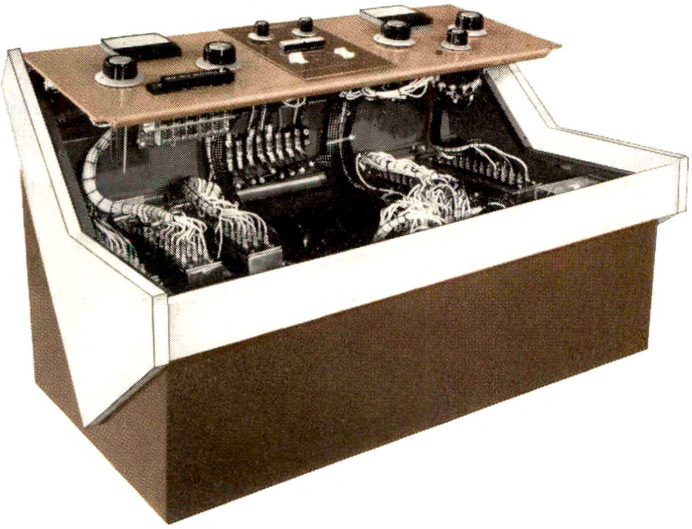

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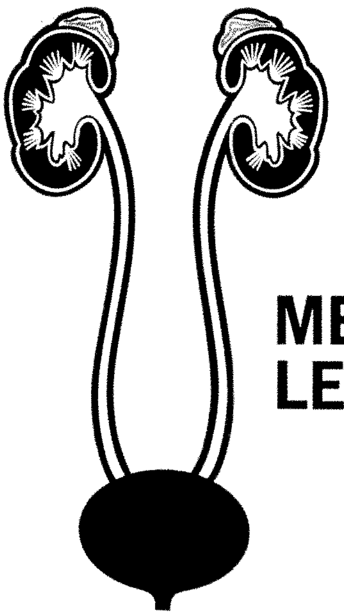

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*Margulis, A. R., and Heinbecker, P.: Am. J. Roentgenol. *86*:103 (July) 1961.

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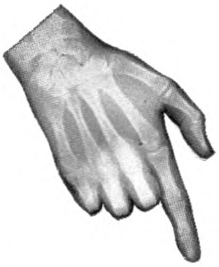

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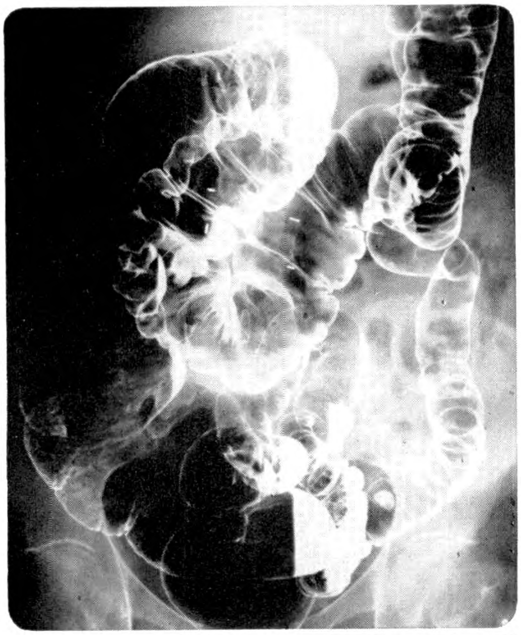

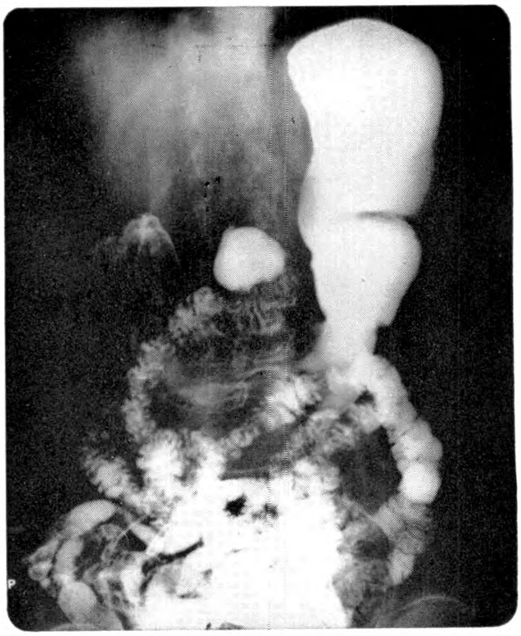

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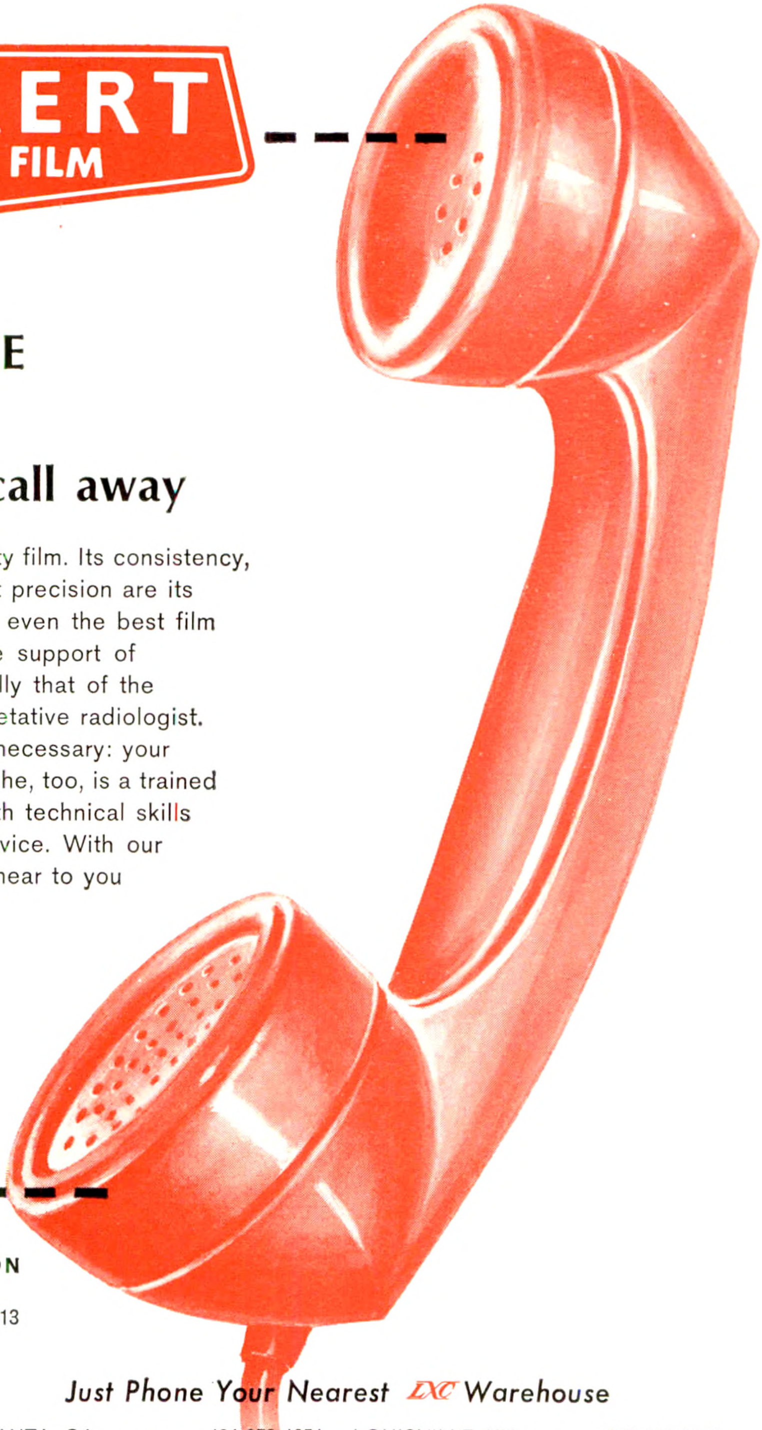

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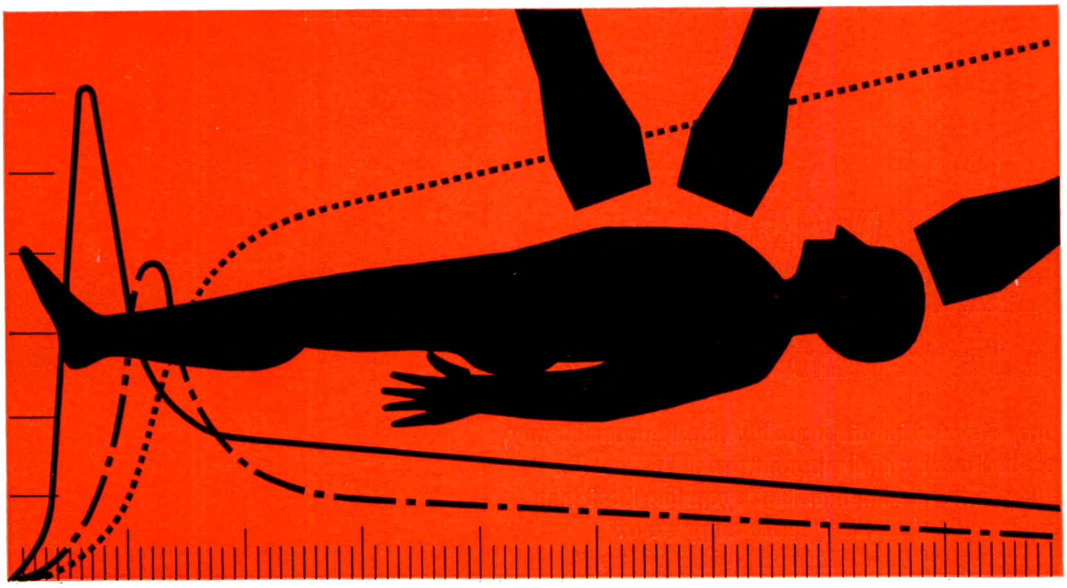

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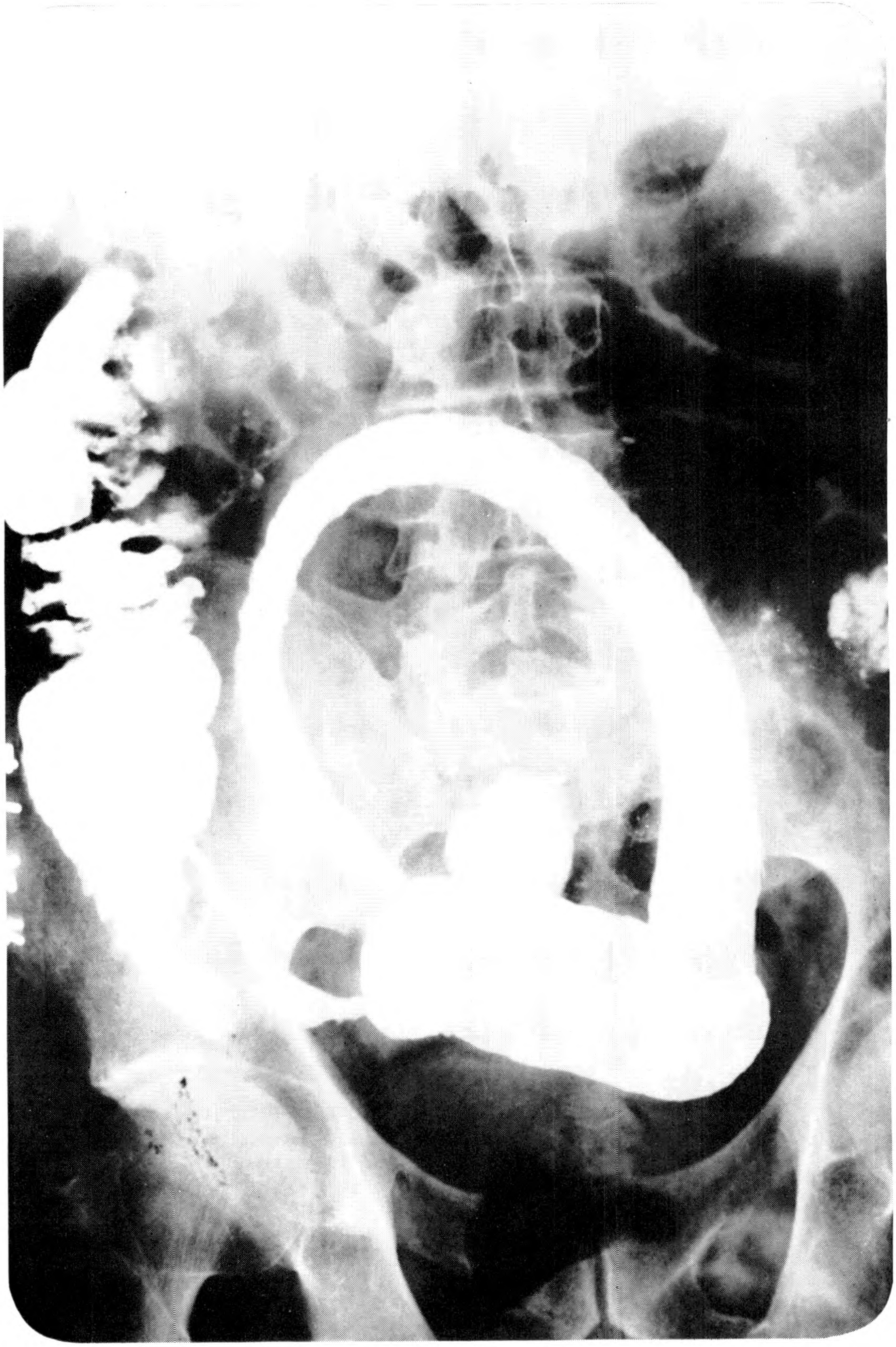

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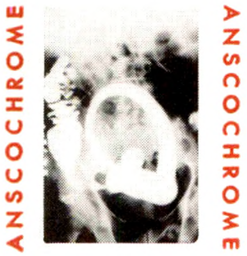

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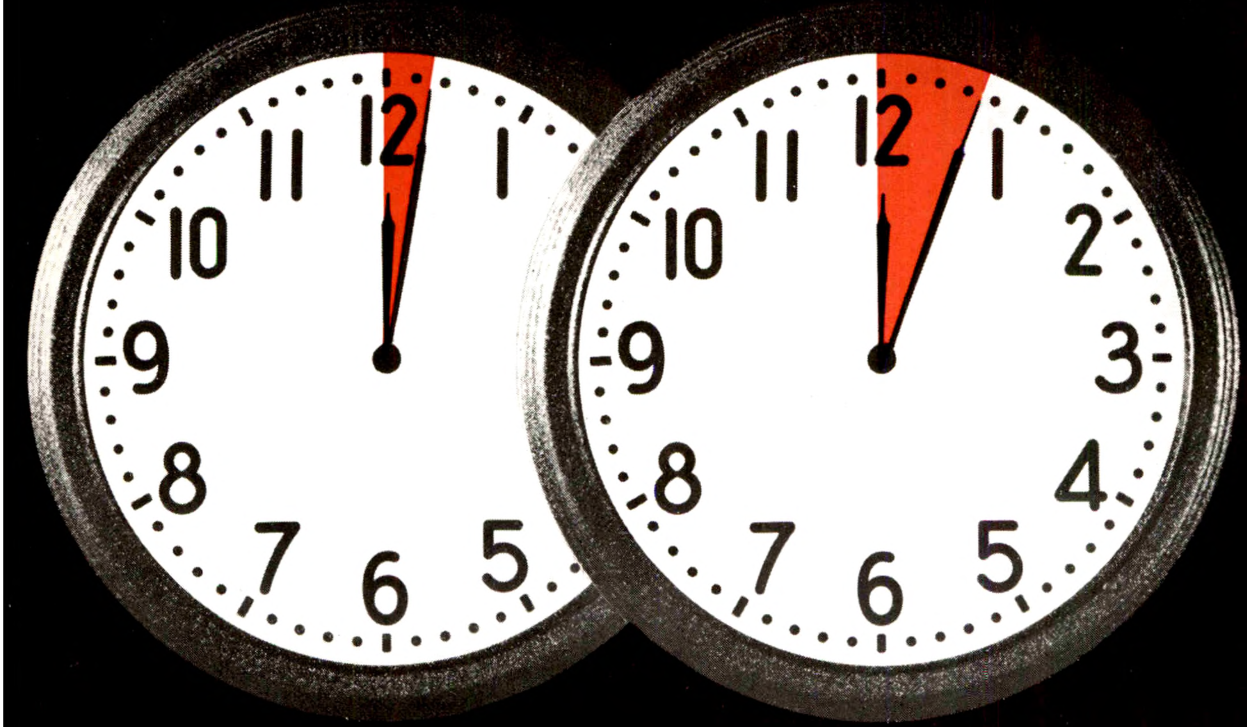

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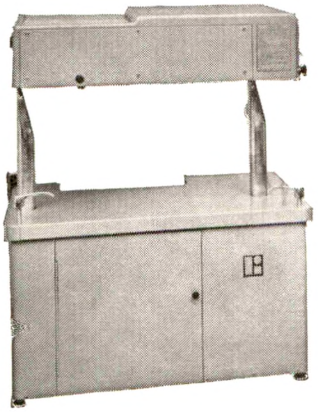

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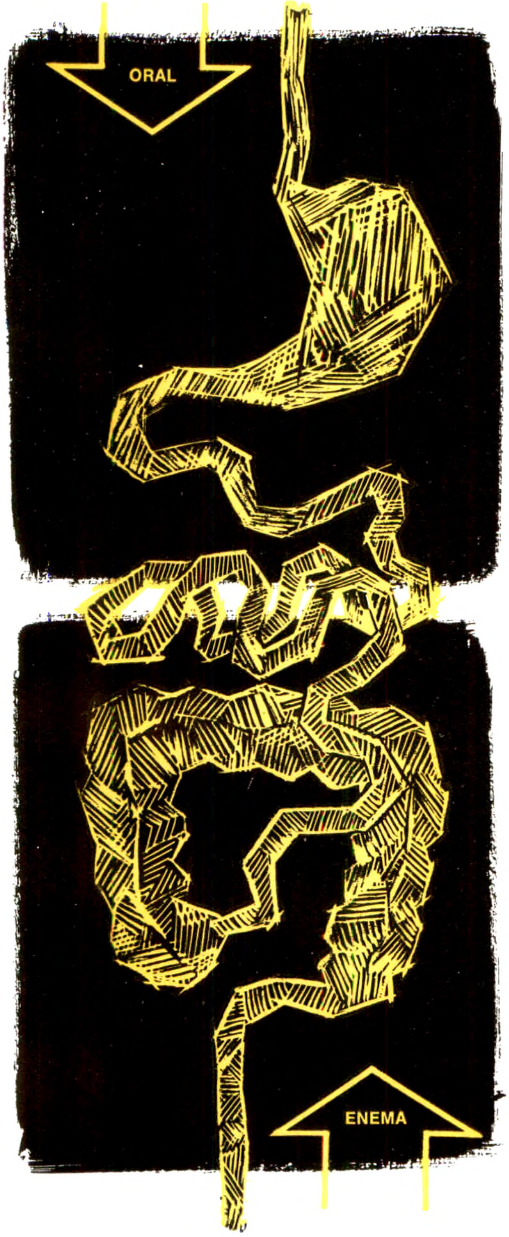

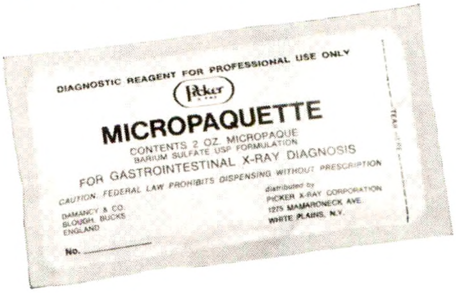
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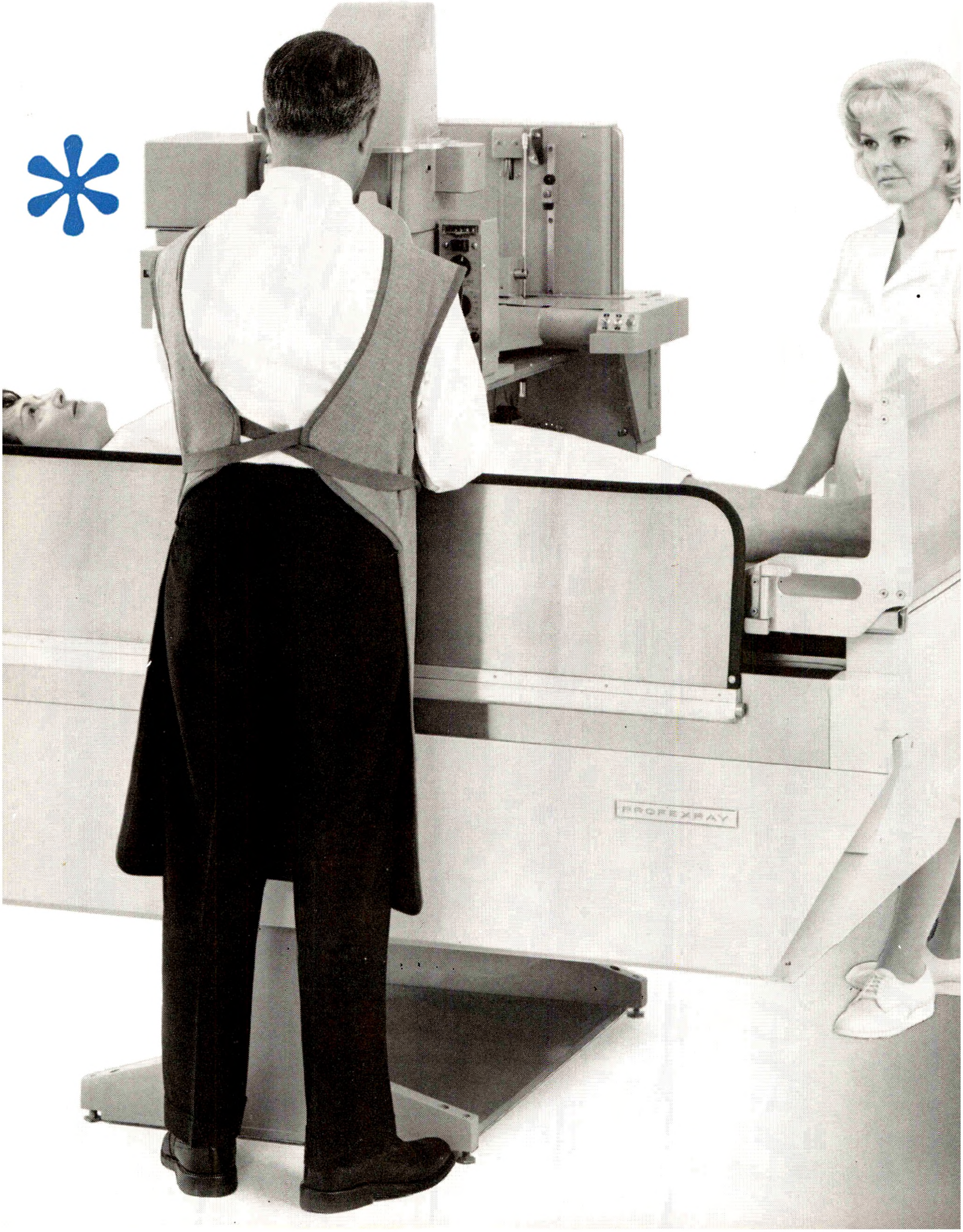
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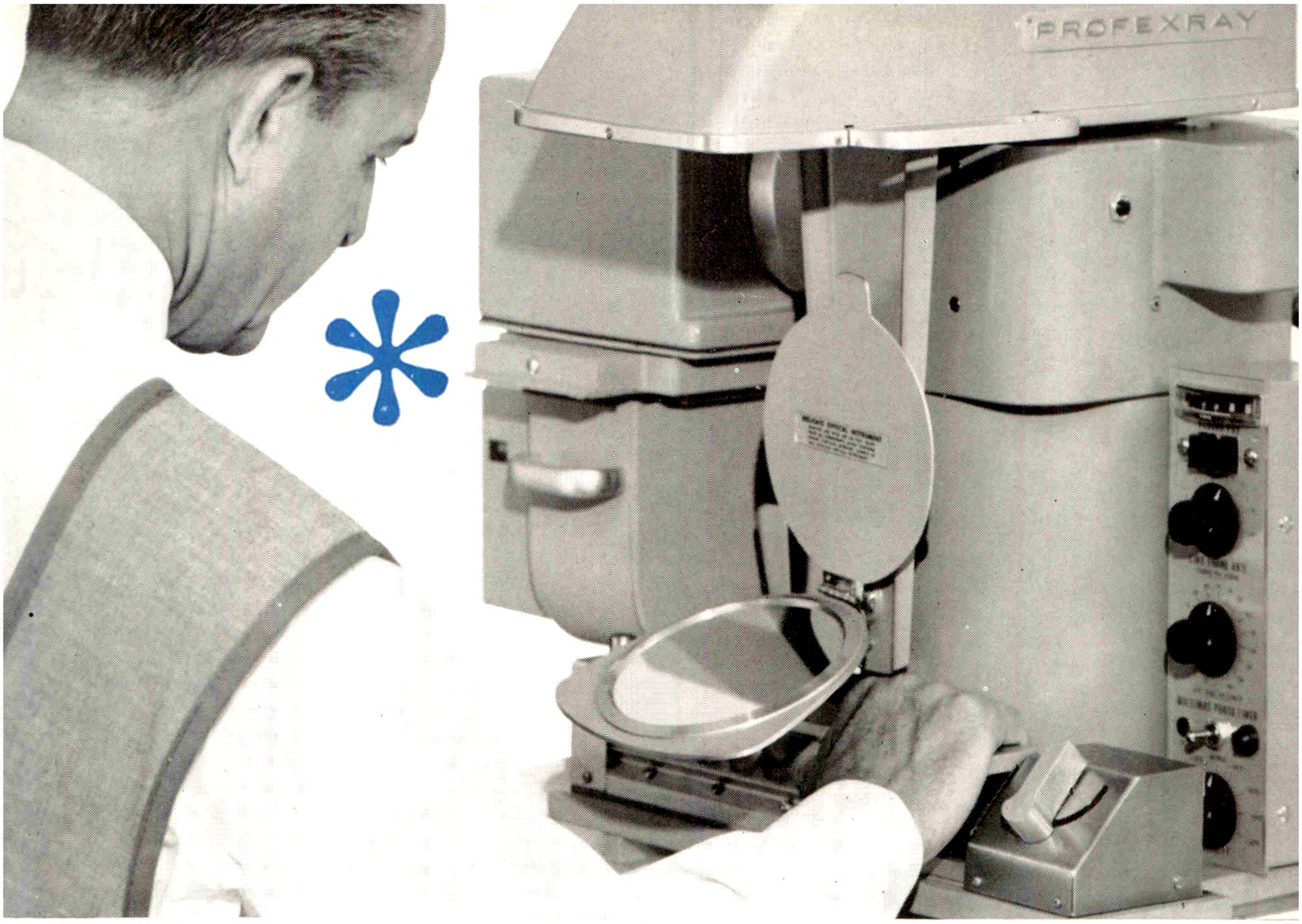

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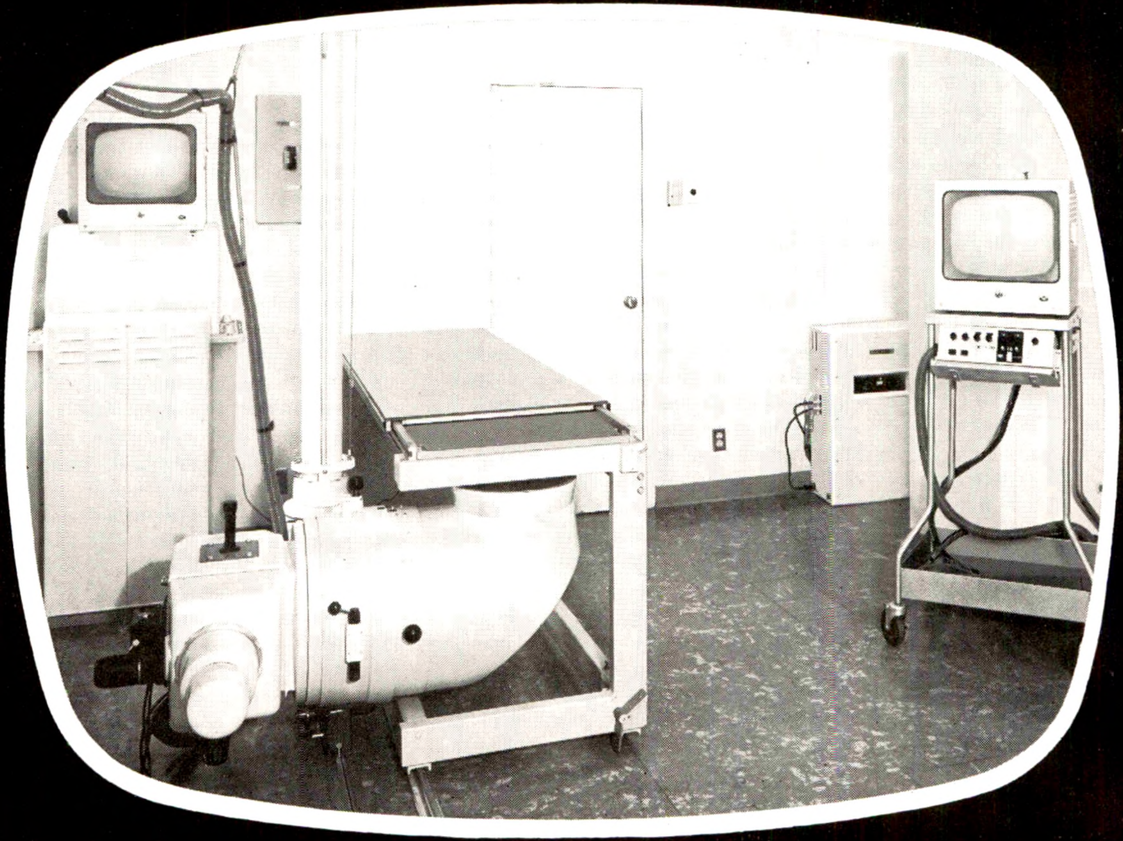

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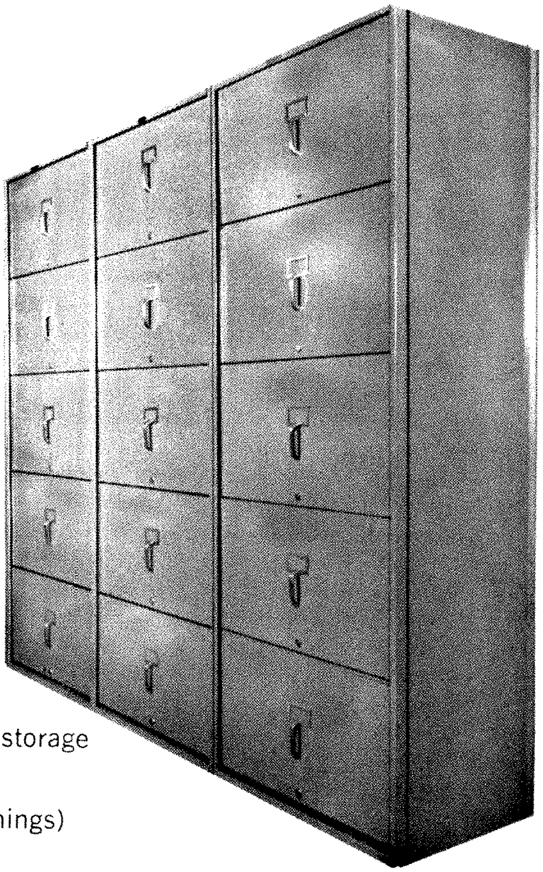

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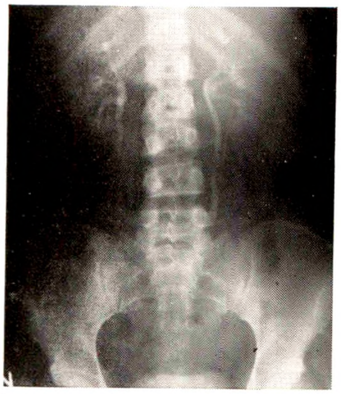

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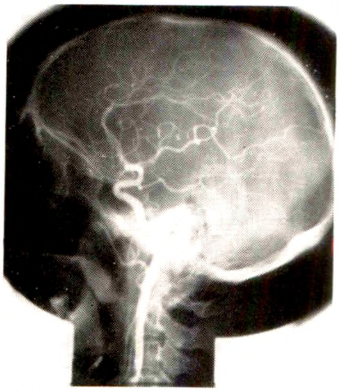

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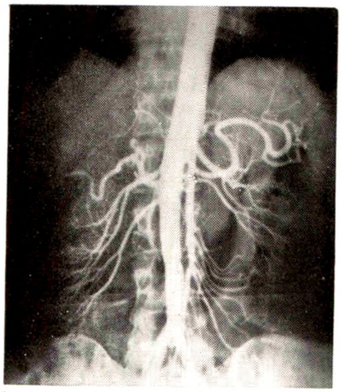

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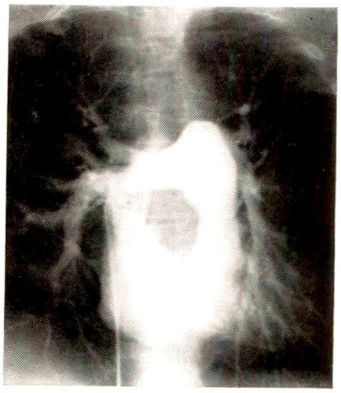

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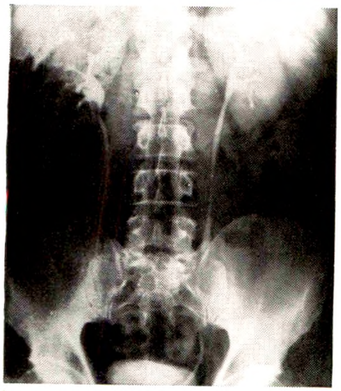

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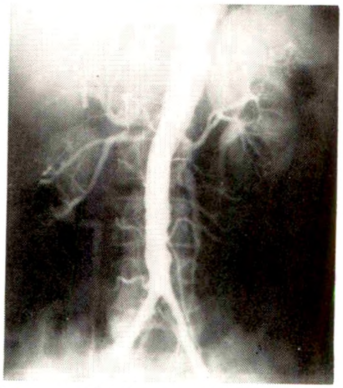

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Contraindications: For all three media, caution is required in cases involving severe systemic disease. In patients with advanced renal disease, use with caution and then only when need for examination dictates. In patients with a history of allergy or those exhibiting a positive reaction to the sensitivity test, drugs and facilities for emergency use must be readily available.

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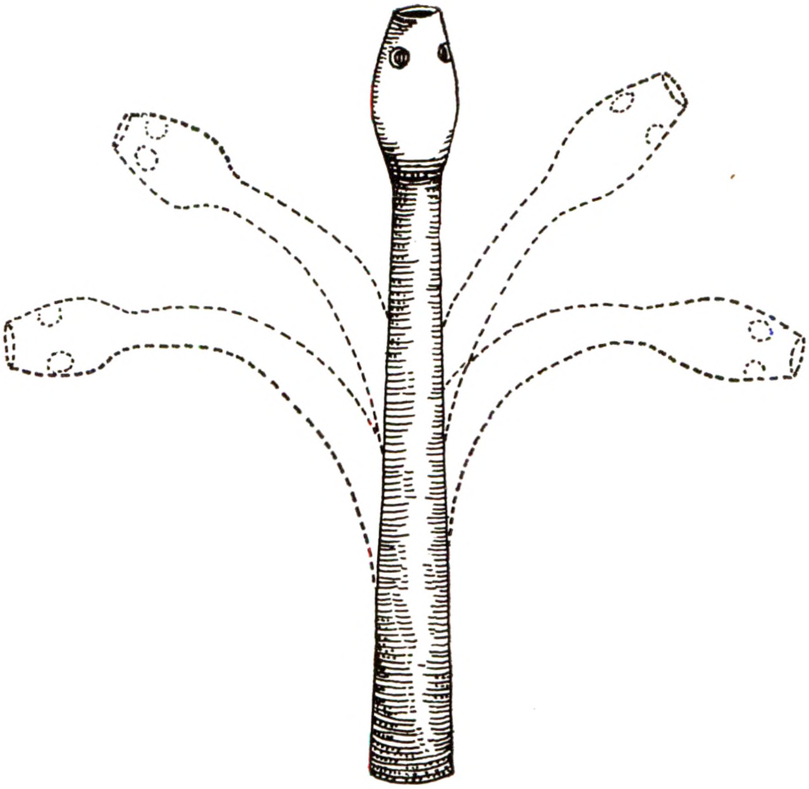

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Bibliography 1. Meyers, P. H.: J.A.M.A. 173: 1589, August 6, 1960. 2. Nathan, M. H.: Am. J. Roentgenol., Rad. Ther. and Nuclear Med. 81: 650, 1959. 3. Steinbach, H. L., et al.: J.A.M.A. 174: 1207, Oct. 29, 1960 4. Pochaczevsky, R., et al.: Radiology 77: 831, (Nov.) 1961.

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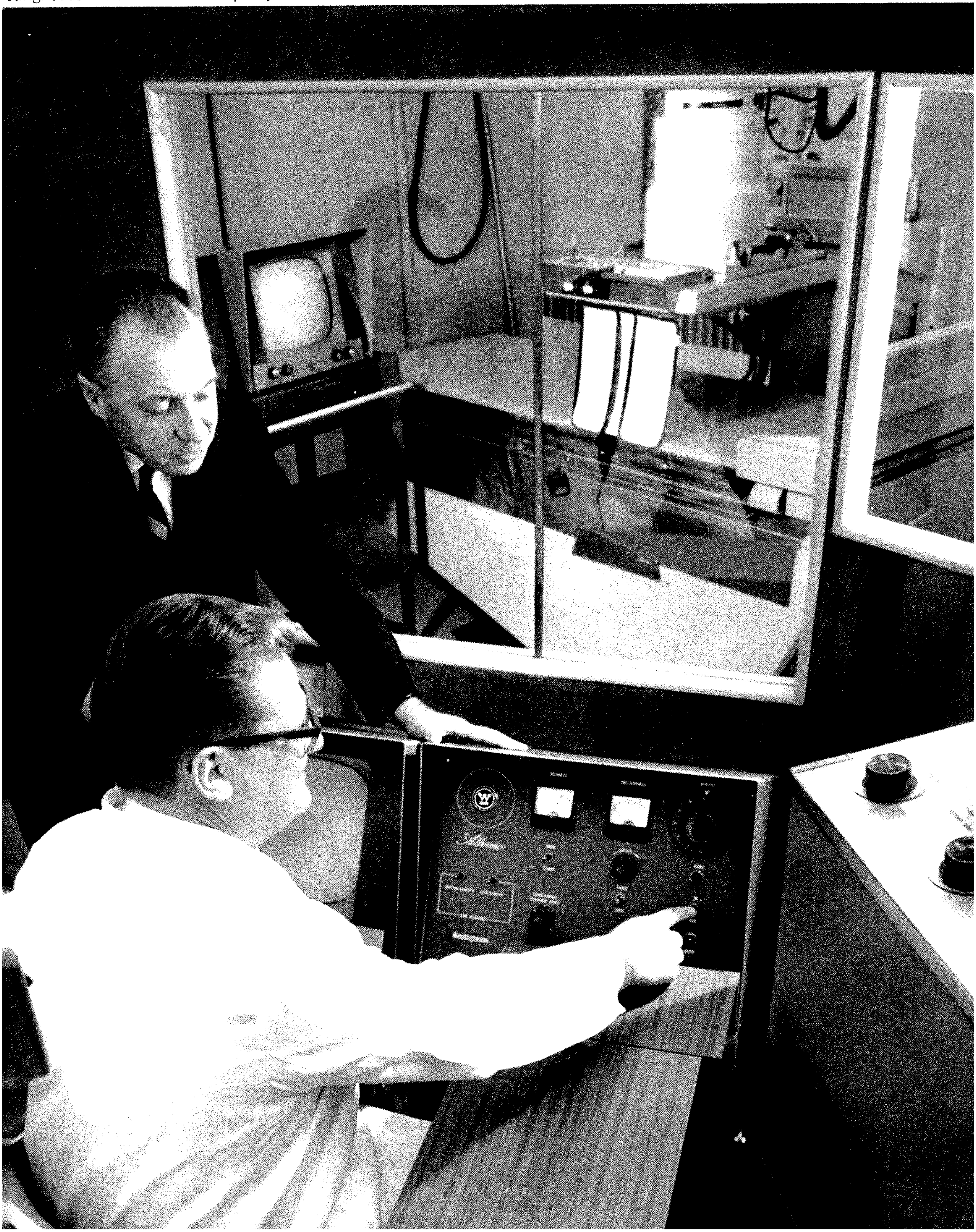

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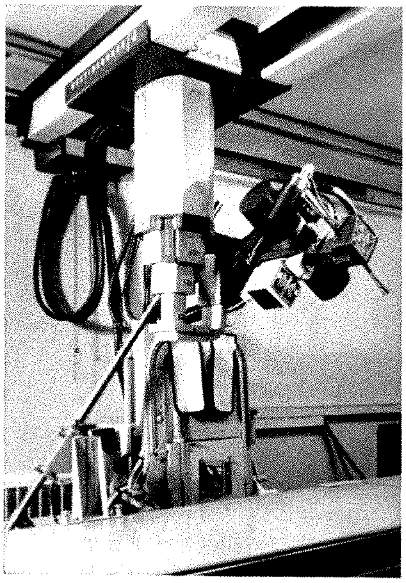

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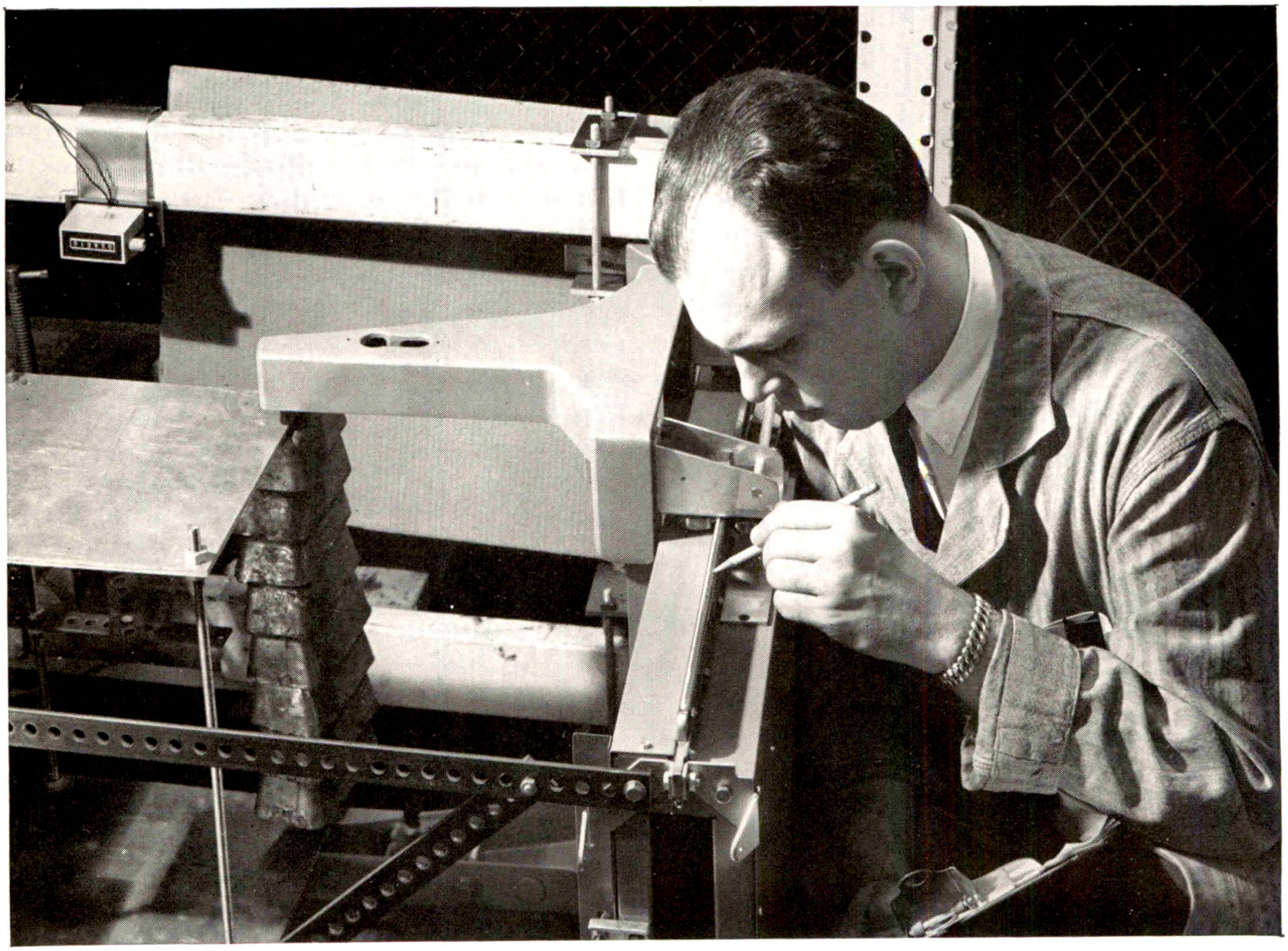

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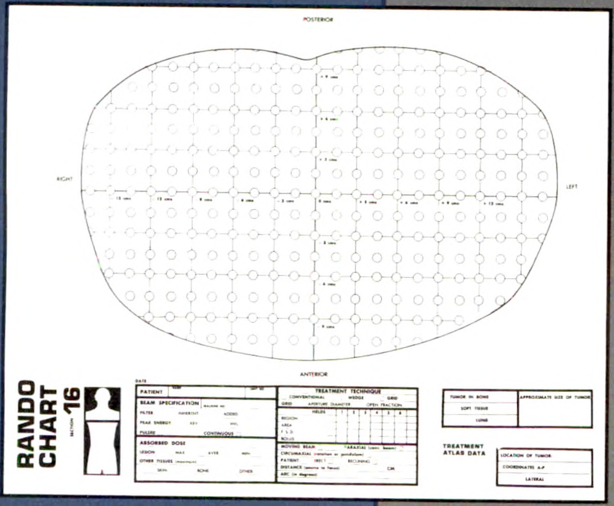

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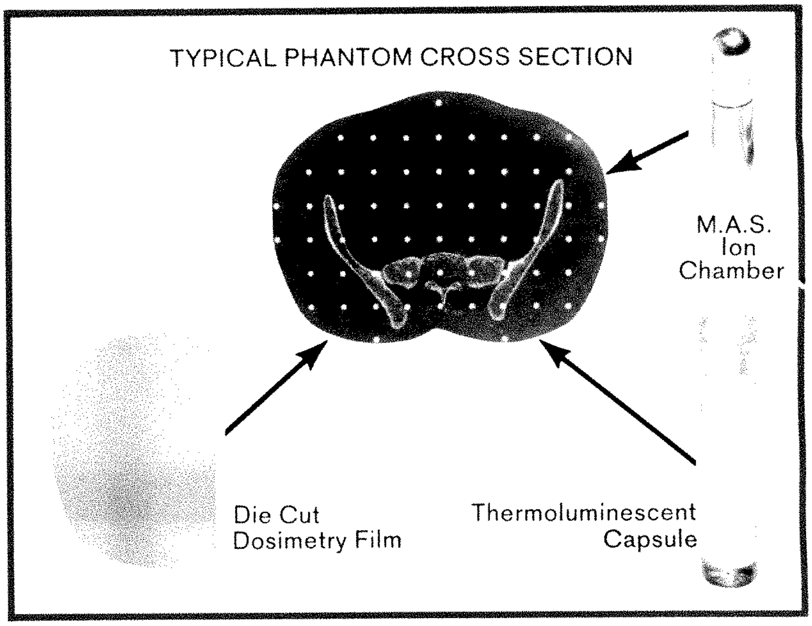

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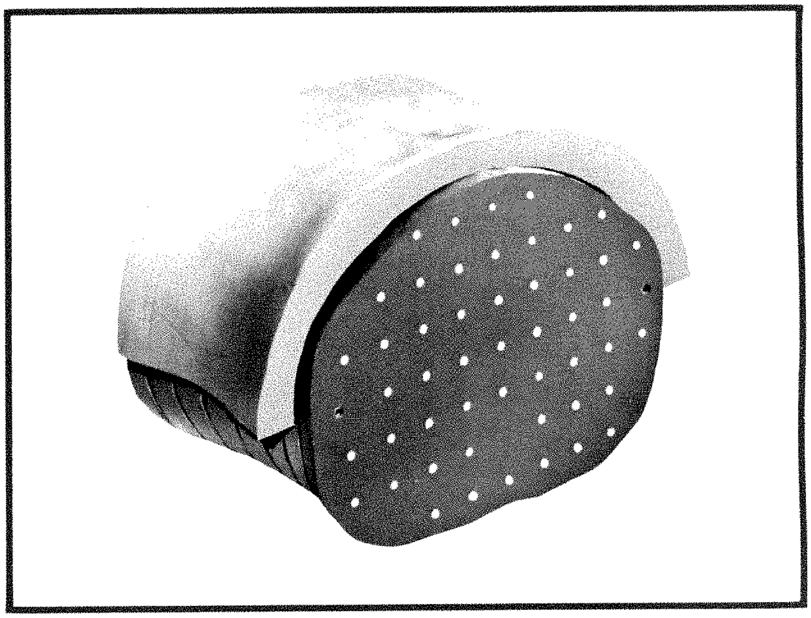

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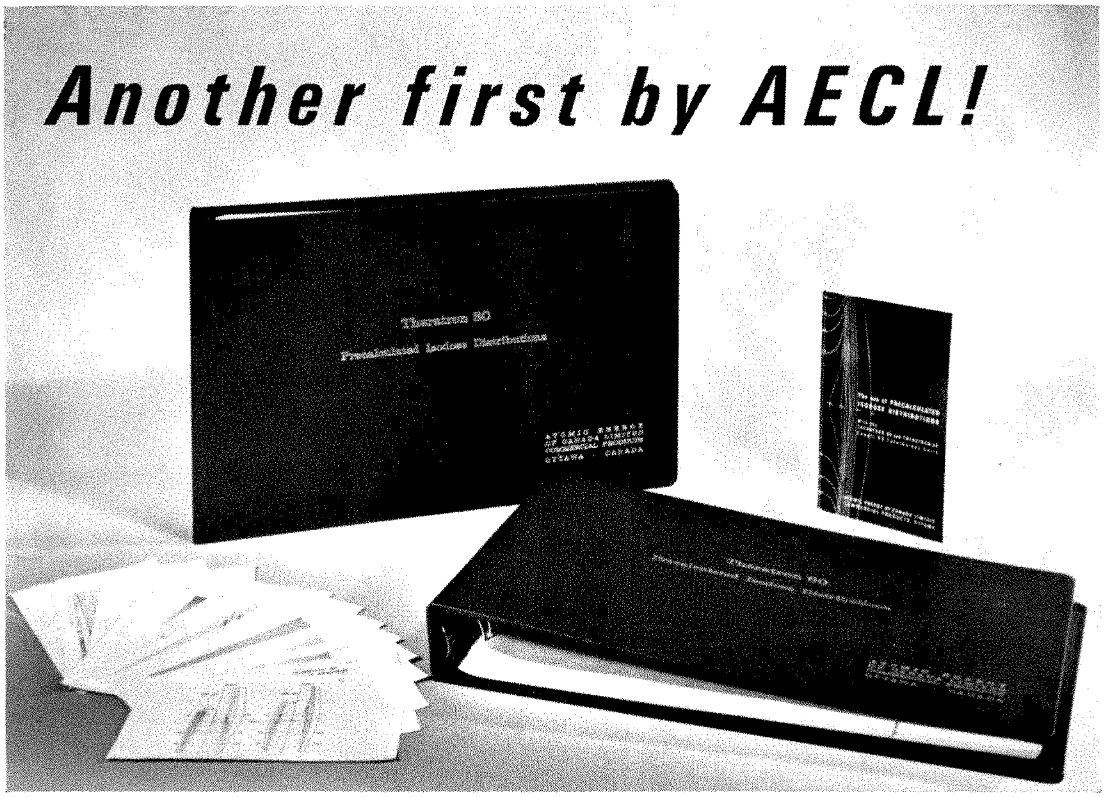

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References: 1. Baker, H. L., Jr., and Hodgson, J. R. (Mayo Clinic and Mayo Foundation): Further studies on the accuracy of oral cholecystography, *Radiology 74*:239, Feb., 1960. 2. Alderson, D. A. (King's College Hosp., London): The reliability of Telepaque cholecystography, *Brit. J. Surg. 47*:655, May, 1960. 3. Rogers, J. V., Jr., Bonmati, J., Leigh, T. F., and Gay, B. B., Jr. (Emory University Hospital, Atlanta): Correlation of radiographic and surgical findings in gallbladder disease, *J.M.A. Georgia 42*:489, Nov., 1953. 4. Tillinghast, A. J. (New York); X-ray diagnosis in disorders of the biliary tract, *Rev. Gastroenterol. 20*:750, Oct., 1953. 5. Mauthe, H. (St. Agnes Hosp., Fond du Lac, Wis.): The accuracy of X-ray examination of the gallbladder, *Wisconsin M.J. 53*:473, Sept., 1954. 6. Garland, L. H. (San Francisco): New techniques in cholecystography, *Minnesota Med. 39*:433, July, 1956. 7. Whitehouse, W. M. (University Hospital, Ann Arbor, Mich.): Correlation of surgical pathology with Telepaque cholecystography in doses of two grams, *Surg., Gynec. & Obst. 100*:211, Feb. 1955. 8. Dietz, M. W., and Litton, L. D. (Springfield, Mo.): Syllabus of cholecystography, *GP 14*:109, Oct., 1956.

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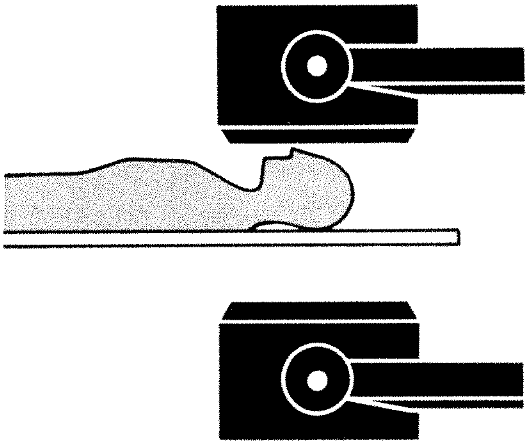

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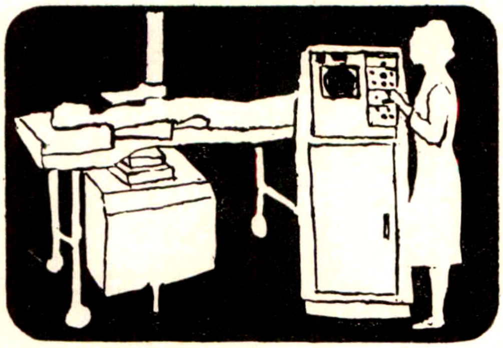
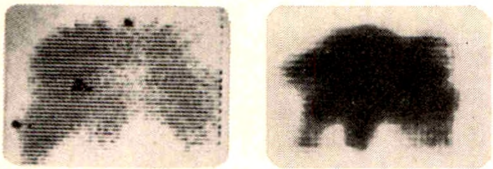
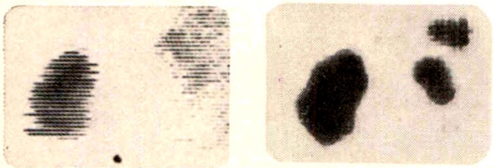




Conventional Scan Time: 35 min. **Autofluorogram Time: 3.2 min.**

At left is a conventional anterior scan compared with a Model 5000 autofluorogram of an abnormal liver showing a hepatomegaly and an abnormal zone of decreased uptake in the lateral margin of the right lobe, which represents a liver metastasis.



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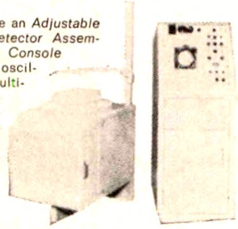

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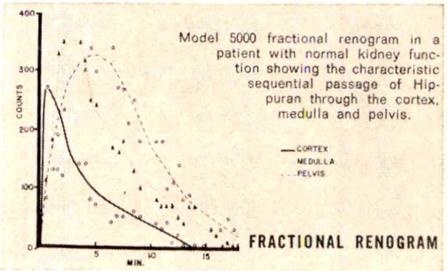

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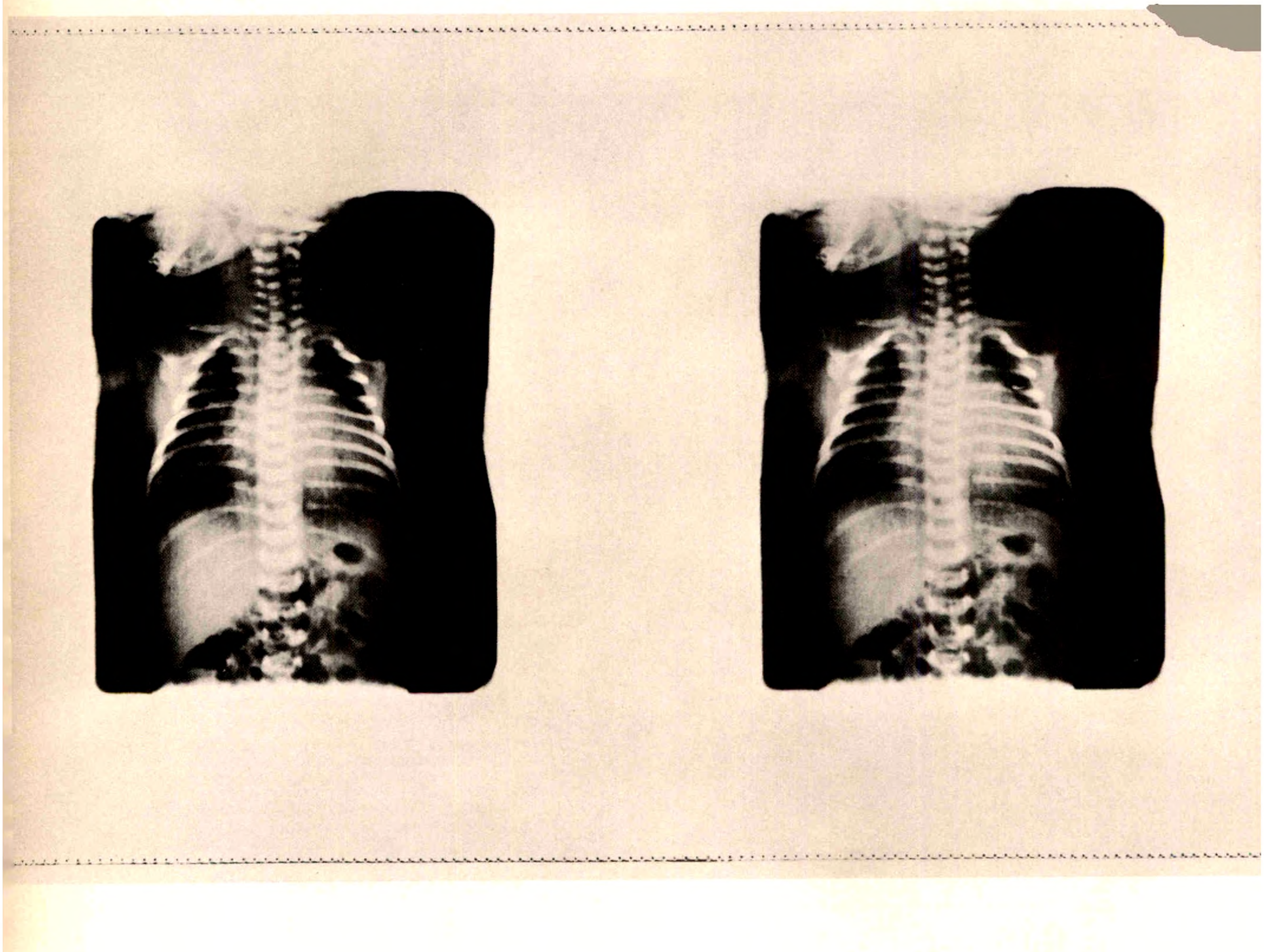

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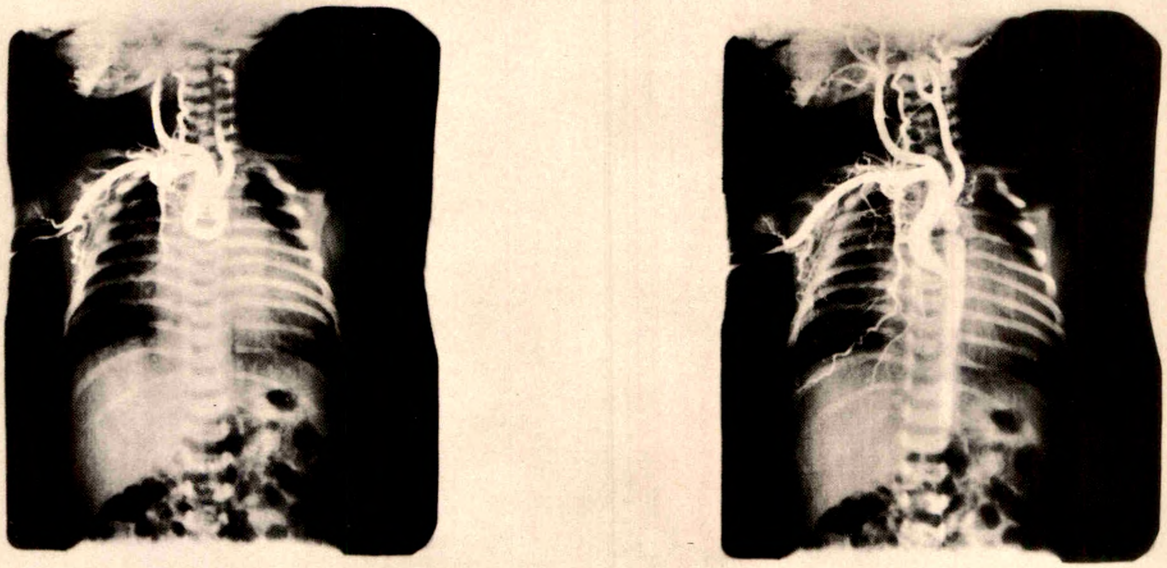

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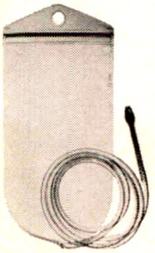

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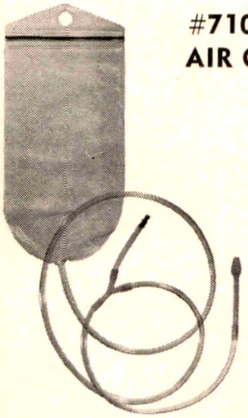

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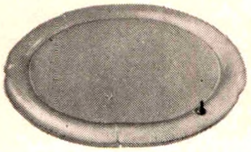

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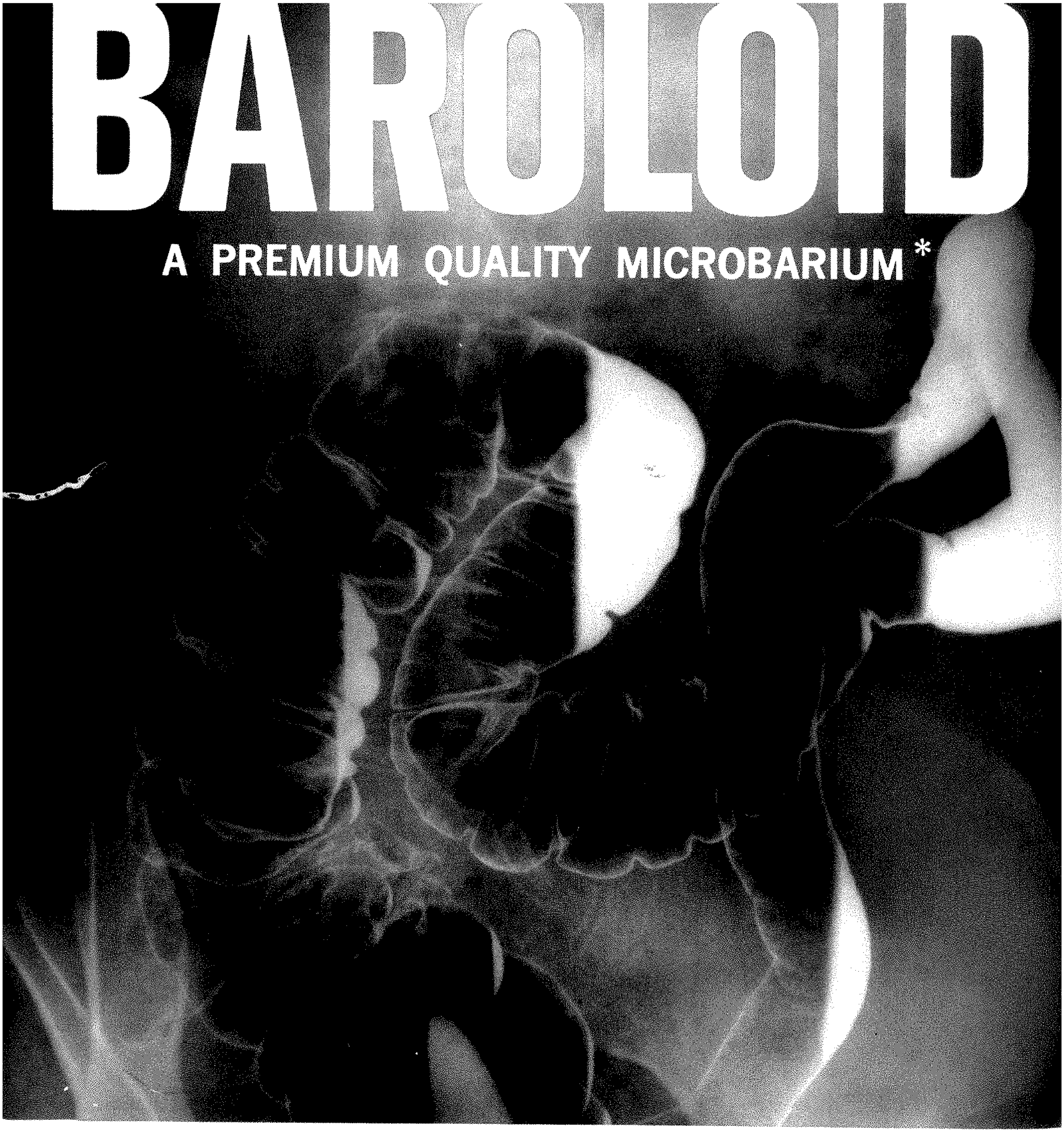

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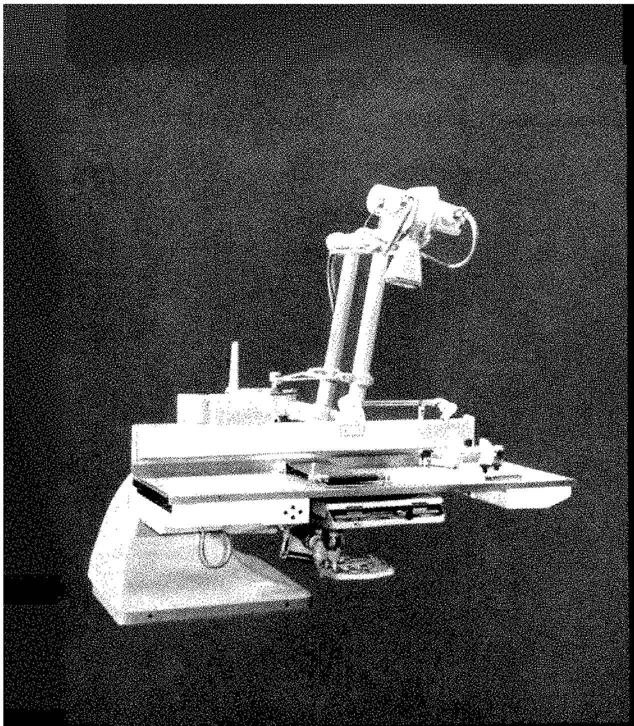

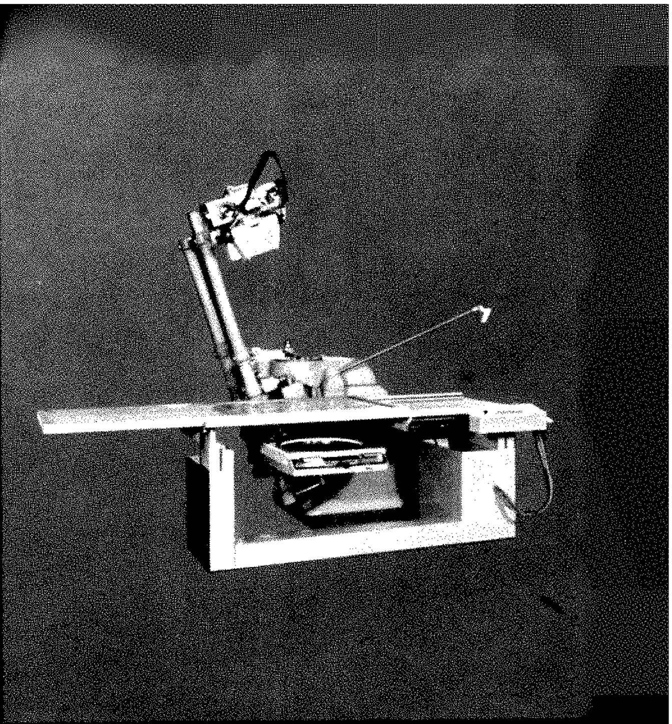

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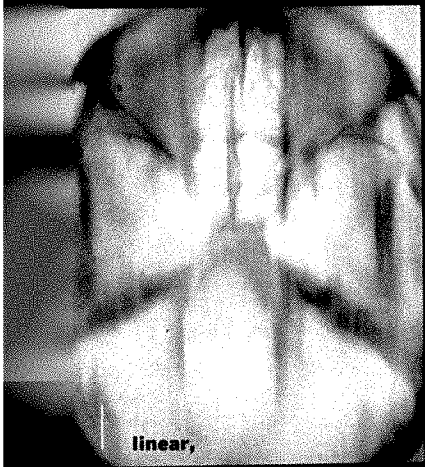

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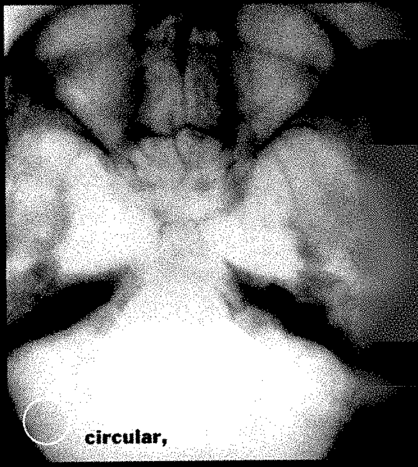

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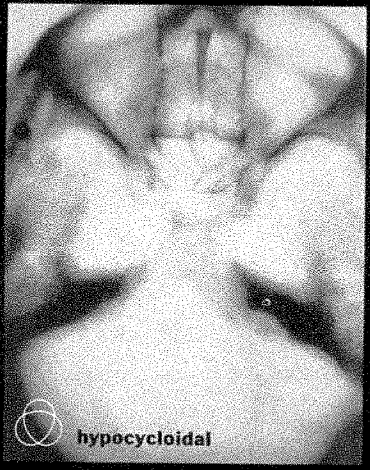

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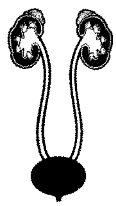

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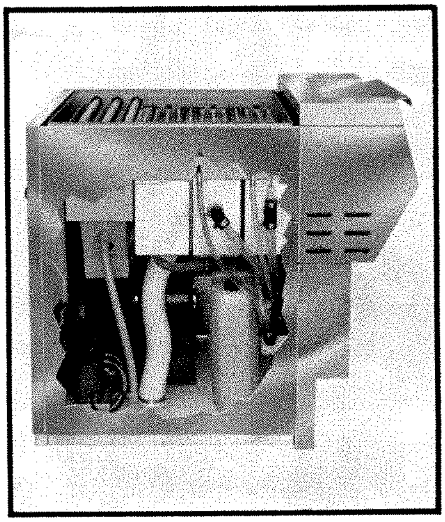

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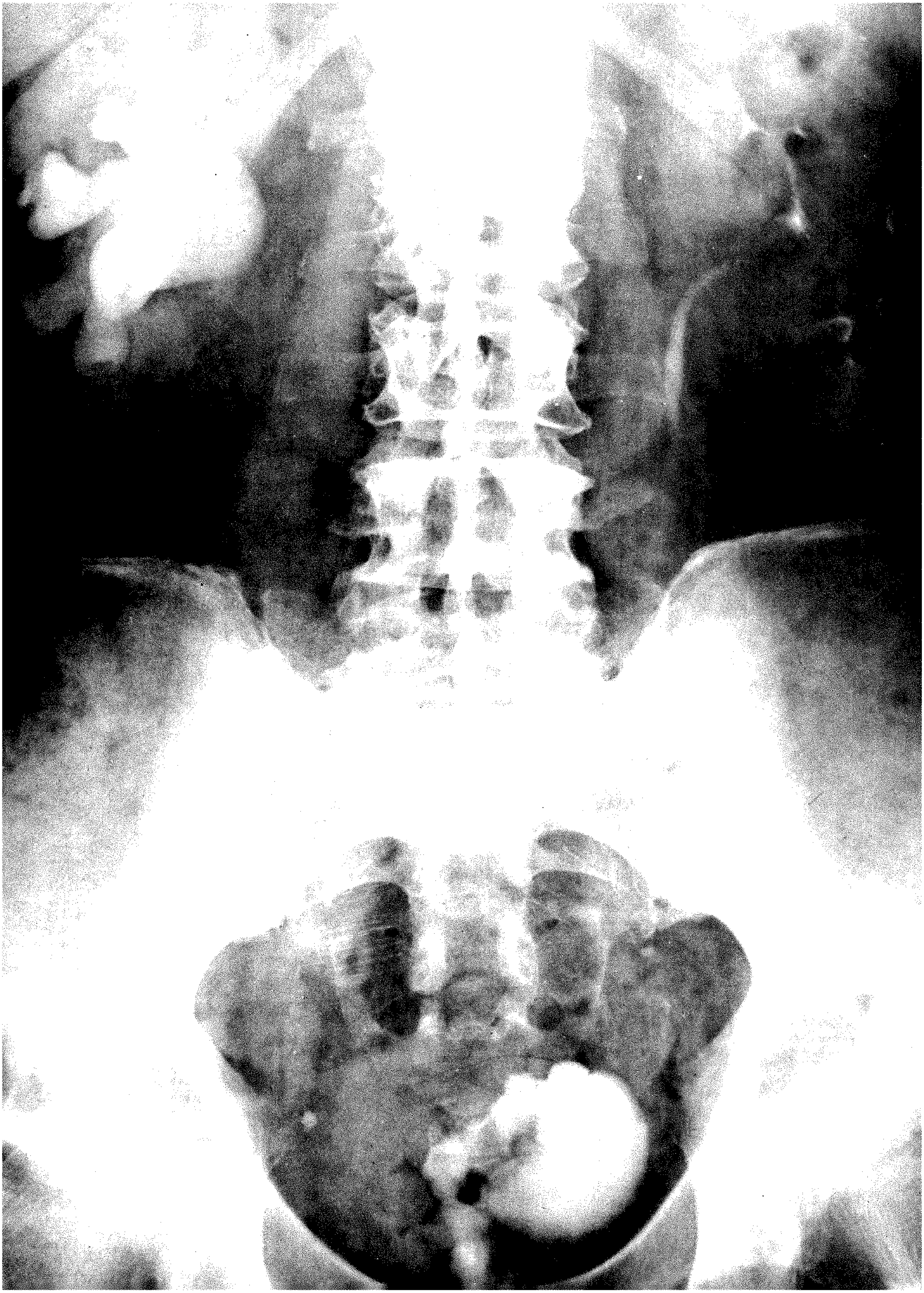

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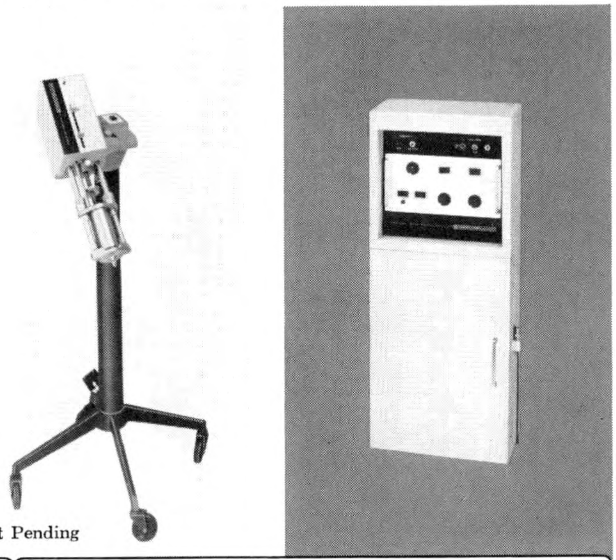

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VOL. 97 MAY, 1966 NO. 1

THE ROENTGENOGRAPHIC FEATURES OF THE KS MUCOPOLYSACCHARIDOSIS OF MORQUIO (MORQUIO-BRAILSFORD'S DISEASE)*

By LEONARD O. LANGER, JR., M.D., *and* LEWIS S. CAREY, M.D.
MINNEAPOLIS, MINNESOTA

MORQUIO'S disease, or to be historically correct, Morquio-Brailsford's disease, is a disproportional dwarf condition independently described in 1929 by Morquio,[16,18] a pediatrician in Uruguay, and by Brailsford,[2] a radiologist in England. This condition is also known as chondro-osteo-dystrophy, osteoechondrodystrophy deformans, eccentrochondroplasia, spondyloepiphyseal dysplasia congenita and Morquio-Ullrich's disease. It is hereditary with an autosomal recessive mode of transmission.

Confusion concerning the diagnostic criteria for Morquio's disease developed soon after the original articles by Morquio and Brailsford. In Morquio's article in 1935,[17] he mentioned 8 articles by other authors who considered their patients to have Morquio's disease. Only 3 of these articles actually described patients with this condition. Brailsford, in his subsequent publications, included a number of conditions under his term chondro-osteo-dystrophy which were different from his original case. As a result, most dwarfs of the short trunk variety showing roentgenographic evidence of universal platyspondyly have been and are still being classified as Morquio's disease.

On the basis of recent clinical, roentgenographic, and biochemical studies, it has become apparent that (1) Morquio's disease is a specific biochemical disorder characterized by the presence of keratosulfate in the urine; (2) bone changes in this condition are diagnostic and include a specific pattern of universal platyspondyly; (3) corneal opacities and dental abnormalities are a constant feature of this condition; (4) dwarfs with other patterns of universal platyspondyly do not have urinary keratosulfate, corneal opacities or dental abnormalities; (5) platyspondyly, *per se*, is not an adequate criterion for the diagnosis of Morquio's disease; and, (6) an unequivocal roentgenologic diagnosis of Morquio's disease can be made at an early age (1 year) before typical clinical deformity is manifested.

---

* Aided by Grants from the National Foundation, Wallace Cole Orthopedic Fund, and U. S. Public Health Service AMI-F2 19,212 and HE-O 6314.

MATERIAL

This article deals with the diagnostic roentgenographic features of Morquio's disease. Our analysis is based on the following: I. Roentgenographic, clinical, and laboratory studies on 23 patients having universal platyspondyly who were personally seen by one of the authors (LOL). Roentgenographic studies were reviewed on 6 additional patients with universal platyspondyly. II. Review of cases in English literature designated as spondyloepiphyseal dysplasia congenita (Morquio's disease) by Rubin[21] in his textbook, "Dynamic Classification of Bone Dysplasias." III. Review of all legitimate cases of Morquio's disease in the world literature according to Maroteaux, Lamy and Foucher[14] in their recent article on this condition. IV. Review of English language cases reported as Morquio's disease since the above two sources and through 1964.

I. PERSONAL SERIES

Of the 23 patients with universal platyspondyly studied at the University of Minnesota, 10 had roentgenographic bone changes identical to those of Morquio's patients and the first patient of Brailsford. All 10 of these patients showed fine corneal opacities, frequently visualized only on slit lamp examination. The 8 patients who had their original teeth showed a very fine enamel layer with sharp pointed cusps. This was present in both the deciduous and permanent teeth and predisposed to caries formation. Our 2 oldest patients had had all their teeth removed because of marked caries. The 5 patients in whom technically satisfactory paper chromatography of the urine was accomplished, showed keratosulfate, an abnormal urinary mucopolysaccharide. The other 13 patients in our series did not have corneal opacities, dental abnormalities, or abnormal mucopolysac-

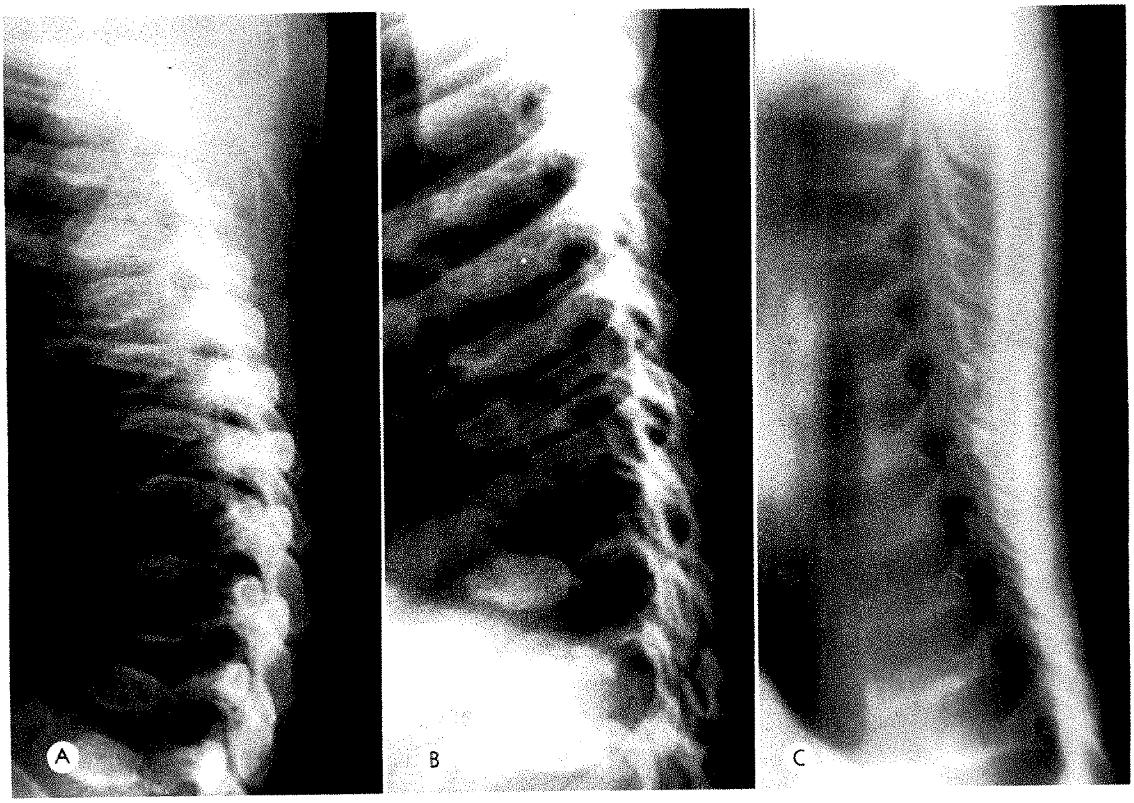

Fig. 1. (*A*) *Young child.* Oval shaped bodies. (*B*) *Older child.* Central "tongue." (*C*) *Adult.* Flattened rectangular shaped bodies.

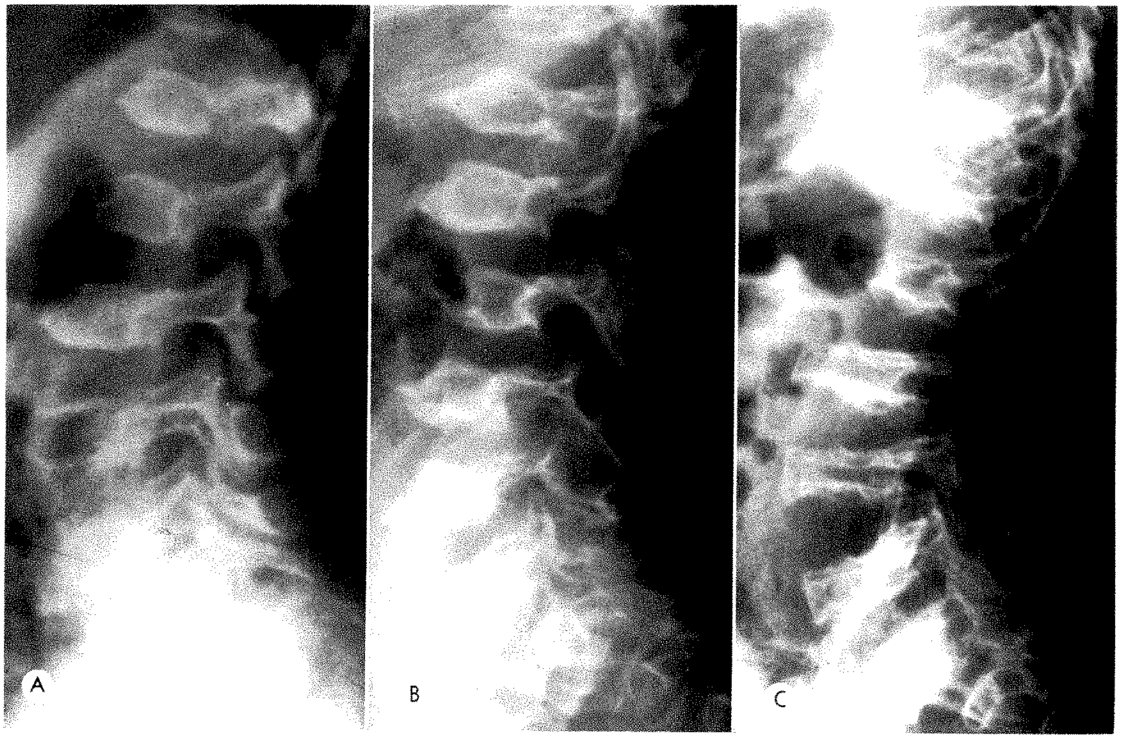

Fig. 2. (*A*) *Young child.* Hook-shaped bodies. (*B*) *Older child.* Further ossification anteriorly. (*C*) *Adult.* Flattened rectangular shape in some bodies.

charide in the urine. They had different patterns of universal platyspondyly from that of Morquio and Brailsford's original cases. It seems clear that these latter patients do not have Morquio's disease, although they have conditions which have been diagnosed as Morquio's disease in the medical literature.

Roentgenographic studies in 6 other patients with Morquio's disease in which teeth changes could be evaluated were reviewed. The ages of personally examined Morquio disease patients range from 15 months to 52 years. The longest serial study covered 22 years. The longest serial study beginning before the age of 2 years covered a 14 year period.

The diagnostic roentgenographic changes of Morquio's disease are seen in the teeth, spine, pelvis, hands and wrists. There is flattening of all of the vertebral bodies, hence the descriptive term, universal platyspondyly, as has been emphasized by all authors. However, this flattening has a specific pattern and, as is true in the other bones of the body, the appearance varies with the age of the individual.

ROENTGENOGRAPHIC FEATURES IN THE YOUNG CHILD

In early childhood, the flattening of the thoracic vertebrae is not marked. In the lateral projection, the bodies have an oval shape (Fig. 1*A*). In the thoracolumbar junction and in the lumbar region, the bodies have a defect in ossification in their anterior superior aspect (Fig. 2*A*). The odontoid process of the second cervical body is mildly hypoplastic (Fig. 3*A*). The pelvis in the young child shows a relatively long pelvic configuration. There is some flaring of the iliac wings and deficient ossification of the superior aspects of the acetabula with mild coxa valga deformity. The femoral necks are heavy without the normal constriction at their mid point. The capital femoral epiphyses are normal or only slightly flattened at this age (Fig.

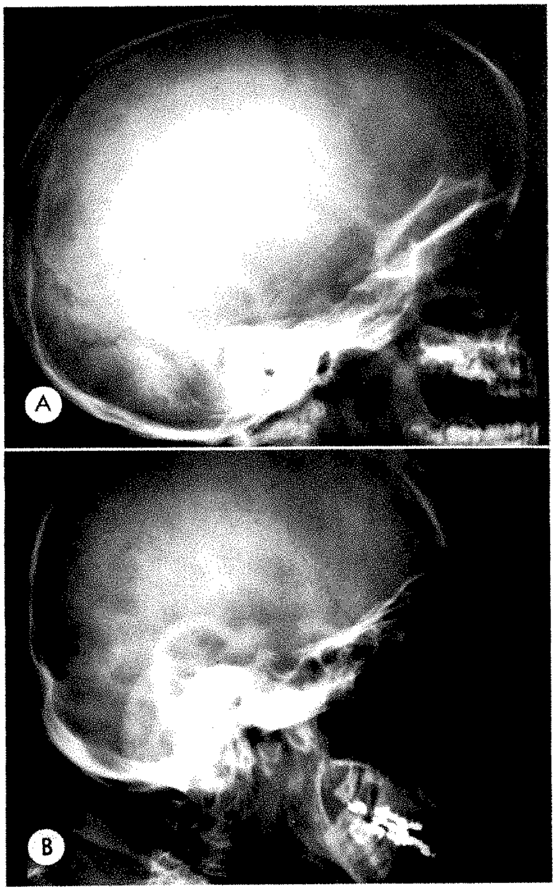

FIG. 3. (*A*) *Young child.* Mild hypoplasia of odontoid; skull normal. (*B*) *Adult.* Concavity of condylar heads; skull normal.

6*A*). The hands show conical bases of the metacarpals 2 through 5. There is some normal constriction of the metacarpal shafts. A normal number of ossified carpal bones are seen at this age. There may be some deformity of the distal ulna with the plane of the growth plate inclined toward the radius (Fig. 7*A*). The enamel layer of the teeth is thin and there are small sharp pointed cusps (Fig. 5).

Other bone changes at this age include abnormal modeling of the humeral, radial, and ulnar shafts and mild dorsal angulation of the humeral necks (Fig. 10*A*). There is a barrel-chest configuration without discrete sternal protrusion (Fig. 9*A*). The skull, mandible (Fig. 3*A*), ribs (Fig. 8*A*), and long tubular bones of the lower extremities (Fig. 11*A*) are normal at this age (Table 1).

ROENTGENOGRAPHIC FEATURES IN THE OLDER CHILD

Evolution of bone changes occurs throughout childhood. The thoracic bodies grow very slowly in height and a central tongue of ossified bone is seen extending anteriorly from the oval shaped body (Fig. 1*B*). There is additional ossification anteriorly in the lumbar vertebrae as well,

TABLE I

DIAGNOSTIC ROENTGENOGRAPHIC FINDINGS IN OUR PATIENTS UNDER THE AGE OF 2 YEARS

	Case 1	Case 2
Teeth Changes	+	NE
Hypoplastic or Absent Odontoid	+ (mild)	NE
Oval-Shaped Thoracic Bodies	+	+ Tongue in lower thoracic bodies
Hook-Shaped Lumbar Bodies	+	+
Flared Ilia	+	+
Long Pelvic Configuration	+	+
Coxa Valga	+ (mild)	+
Deficient Ossification of Superior Acetabula	+	+
Conical Bases of Metacarpals 2–5	+	+
Normal Constriction of Metacarpal Shafts	+	+
Carpal Centers	Normal for age	Normal for age
Deformity of Distal Ulna	+	+

+ = present.
NE = not evaluated.

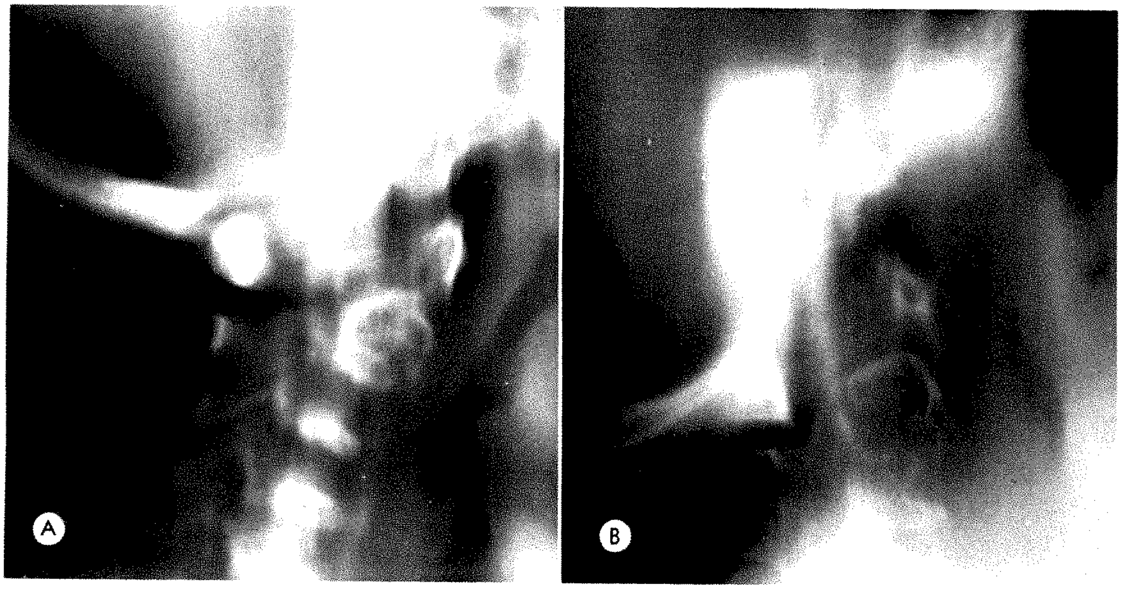

Fig. 4. (*A*) *Older child.* Hypoplasia of odontoid; posterior arches of C1 fit into foramen magnum. (*B*) *Adult.* Absence of odontoid; posterior arches of C1 fit into foramen magnum.

but the hook-shaped configuration persists, particularly in the thoracolumbar junction region. The intervertebral disk spaces are wide (Fig. 2*B*). The odontoid process is hypoplastic or absent (Fig. 4*A*). The pelvis narrows at the level of the acetabula, giving a definite long narrow over-all pelvic configuration. The iliac wings are flared. The superior aspects of the acetabula are poorly ossified. The cleft of the hip joints is wide. The femoral capital epiphyses undergo compression and fragmentation; this is usually first seen between the ages of $3\frac{1}{2}$ and 6 years. The coxa valga deformity is more marked than in early childhood (Fig. 6, *B* and *C*). In the hands, there is additional ossification at the bases of the metacarpals, giving a more normal configuration than in early childhood. The distal metaphyses of the metacarpals 2 through 5 are wider than the adjacent epiphyses. The ossified carpal bones are small and may be reduced in number as compared with the normal standards for the individual's age. Most frequently, the navicular bone does not ossify. The planes of both the distal ulnar and radial growth plates are slanted toward one another (Fig. 7*B*).

Other roentgenographic findings in this age range are broadening of the anterior portions of the ribs and progressive sternal protrusion (Fig. 9*B*). The clavicles are essentially normal in Morquio's disease (Fig. 8*B*) in distinction to the definite clavicular changes which are found in severely affected individuals with the Hurler syndrome. There may be retroplacement of one of the relatively small hook-shaped lumbar vertebrae in relationship to the other vertebrae or anterior displacement of the vertebrae above a hypoplastic hook-shaped vertebra in the thoracolumbar junction region (Fig. 2*B*). At the base of

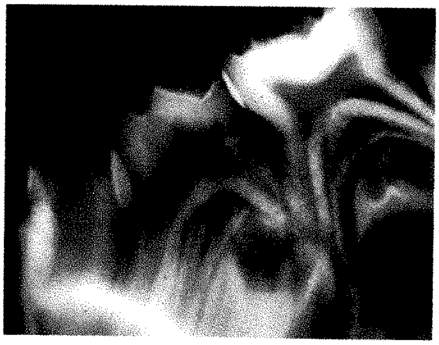

Fig. 5. Thin enamel layer in both deciduous and unerupted permanent teeth; small pointed cusps.

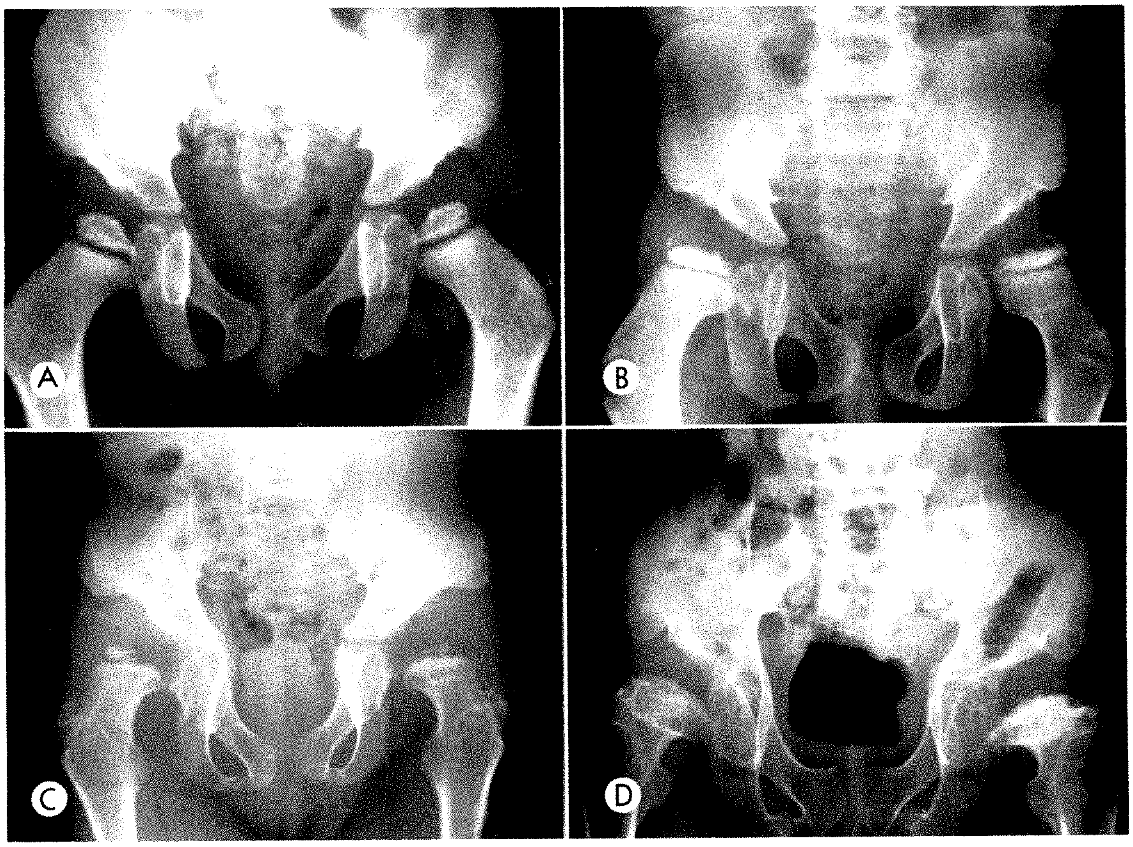

Fig. 6. See text for discussion of pelvic changes. (*A*) *Young child*. Minimal flattening of capital femoral epiphyses. (*B*) *Age 2–6 years*. Beginning of compression of capital femoral epiphyses. (*C*) *Age 6–16 years*. Marked compression and fragmentation of capital femoral epiphyses. (*D*) *Adult*. Disappearance of femoral heads; erosion and widening of femoral necks.

the skull in addition to hypoplasia of the odontoid process, there appears to be excess motion of C1 in relationship to C2 and the posterior arches of C1 fit into the posterior aspect of the foramen magnum (Fig. 4*A*). It is this relationship, rather than modification of the occipital bone, which gives the effective basilar impression in Morquio's disease. The previously enumerated teeth changes are seen in both the deciduous and permanent teeth. In the pelvis, the symphysis pubis is wider than normal (Fig. 6, *A–D*). The shoulder joints are wider than normal. The surgical neck of the humeri shows dorsal angulation and there is some irregularity at the distal end of the humeri. Commonly, the proximal ulnar and radial shafts show widening, particularly in the region of the tuberosity of the radius. The radius and ulna are frequently shorter than normal and the ulna is disproportionately shorter than the radius (Fig. 10*B*). In the lower extremities, the distal femoral metaphyses show some sclerosis along the growth plate. There is increased trabeculation in the lower portion of the femur, sometimes simulating the appearance of the linear seams of cartilage seen in enchondromatosis. These changes are apparently due to stress phenomena. The distal femoral growth plate may be somewhat oblique to the shaft of the femur contributing to the genu valgum deformity. Similar obliquity may be seen in the proximal tibial metaphyseal region. There is frequently less good ossification of the lateral aspect of the proximal tibial epiphysis than of the medial aspect. Separate small ossification centers

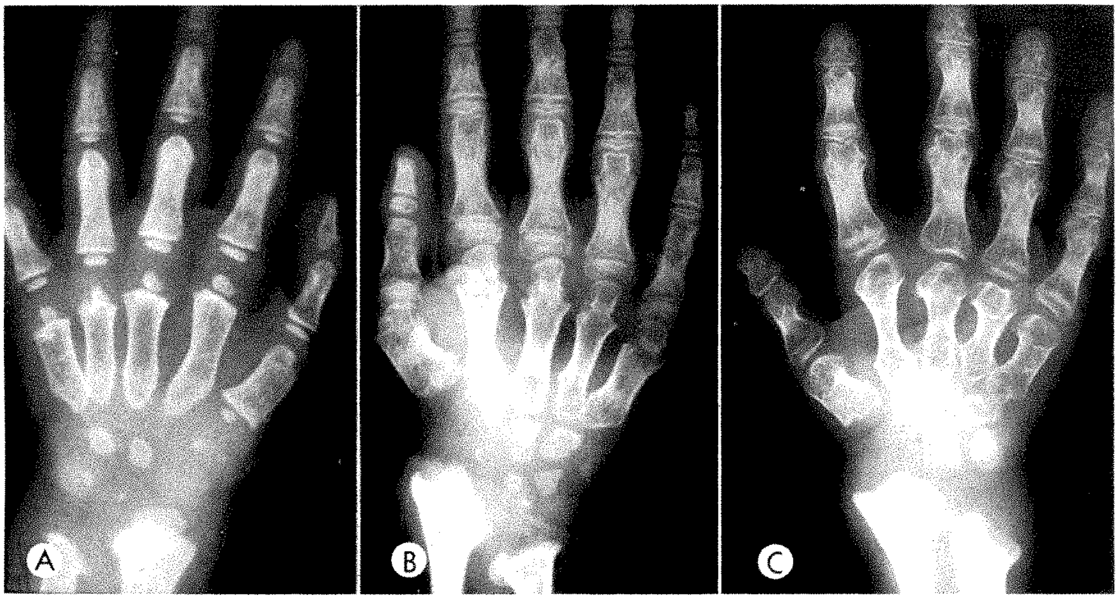

Fig. 7. (*A*) *Young child.* Conical bases of metacarpals 2–5; normal constriction of metacarpal shafts; normal number of ossified carpal bones; obliquity of distal ulnar growth plate. (*B*) *Older child.* Distal metaphyses of metacarpals wider than adjacent epiphyses. Normal constriction of metacarpal shafts; decreased number and small size of ossified carpal bones; obliquity of both distal ulnar and radial growth plates. (*C*) *Adult.* Fusion of growth plates; normal constriction of metacarpal shafts; decreased number of ossified carpal bones; less marked deformity of distal ulna and radius.

are seen on the lateral aspect (Fig. 11*B*). The joint cleft of the knees is wide. The changes in the feet are similar to those in the hands although less marked (Table II and III).

ROENTGENOGRAPHIC FEATURES IN THE ADULT

The anterior portions of many of the vertebrae ossify and give a normal but flattened rectangular configuration on lateral roentgenograms (Fig. 1*C*). There is still an anterior superior osseous defect in one or more bodies in the thoracolumbar junction region. The height of the bodies does not increase from late childhood. The disk spaces remain wide (Fig. 2*C*). The odontoid process is hypoplastic or absent with fitting of the posterior arches of C1 into the foramen magnum (Fig. 4*B*). There appears to be less relative motion of C1 on C2 in flexion and extension roentgenograms at this age. In the older child and adult, there is flattening or actual concavity in the configuration of the condylar head of the mandible (Fig. 3*B*). These changes are the same as those described by Horrigan and Baker[10] in individuals with the conditions of the Hurler syndrome. At this age in addition to the teeth changes previously noted, there are many fillings present or all the teeth have been extracted because of numerous dental caries. In the pelvis, the configuration is the same as described in late childhood. Now complete disappearance of the ossified femoral heads with erosion and widening of the femoral neck occurs. There is not, however, coxa vara. This deformity is never present in Morquio's disease. The joint cleft remains wide unless gross subluxation occurs, and the symphysis pubis is wider than normal (Fig. 6*D*). Roentgenograms of the hand usually show shortening of the metacarpals. The carpal bones are small and may be decreased in number. There may be some restoration of a more normal configuration of the distal radius and ulna. Frequently, there is ulnar deviation of the hand because

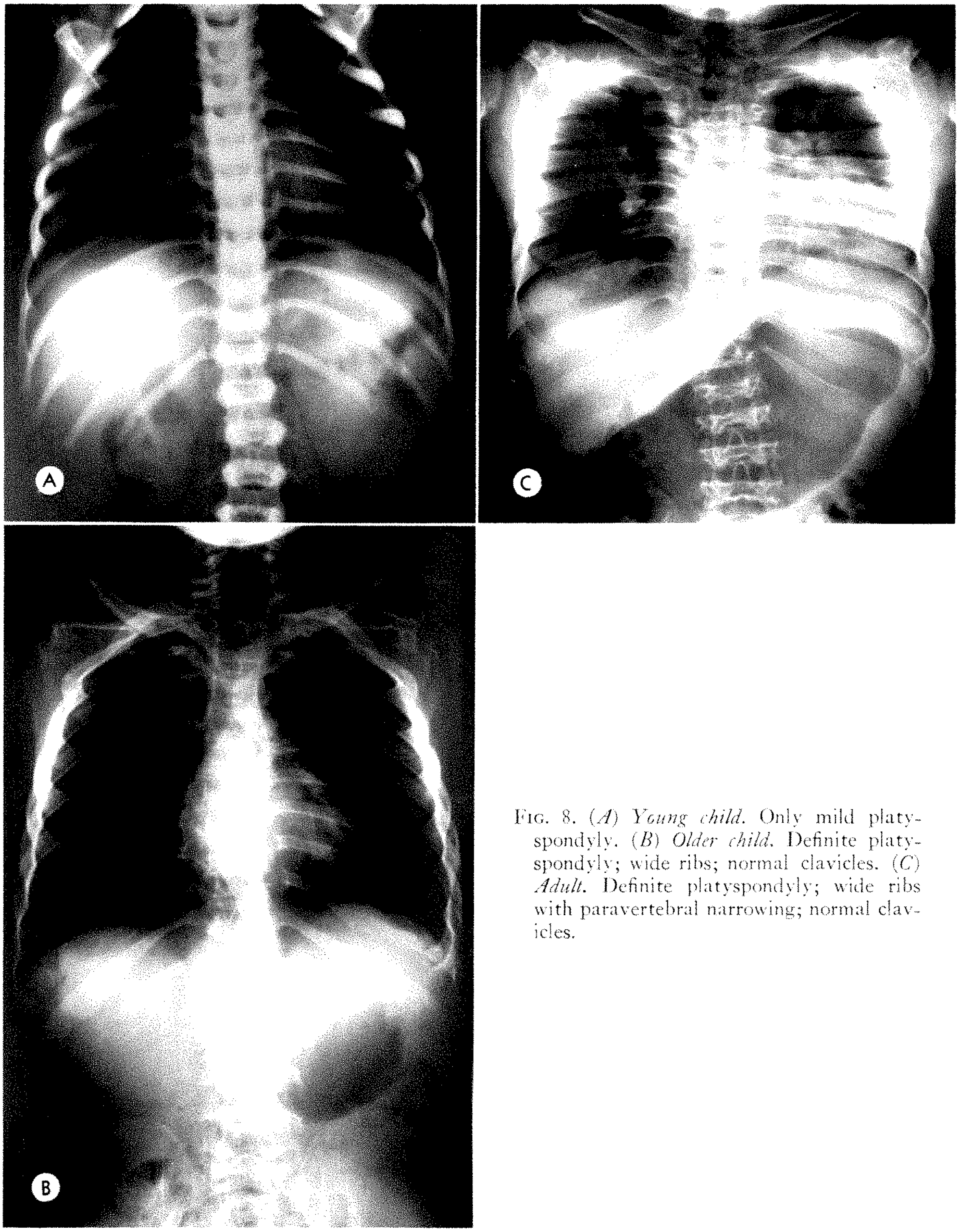

FIG. 8. (*A*) *Young child*. Only mild platyspondyly. (*B*) *Older child*. Definite platyspondyly; wide ribs; normal clavicles. (*C*) *Adult*. Definite platyspondyly; wide ribs with paravertebral narrowing; normal clavicles.

of deformity and shortening of the ulna. Fusion of the epiphyses occurs at a normal age (Fig. 7*C*).

Other roentgenographic findings include widening of the ribs combined with a narrowing in the paravertebral portions, particularly in the lower ribs (Fig. 8*C*). The sternum is thick; there is absence of bony

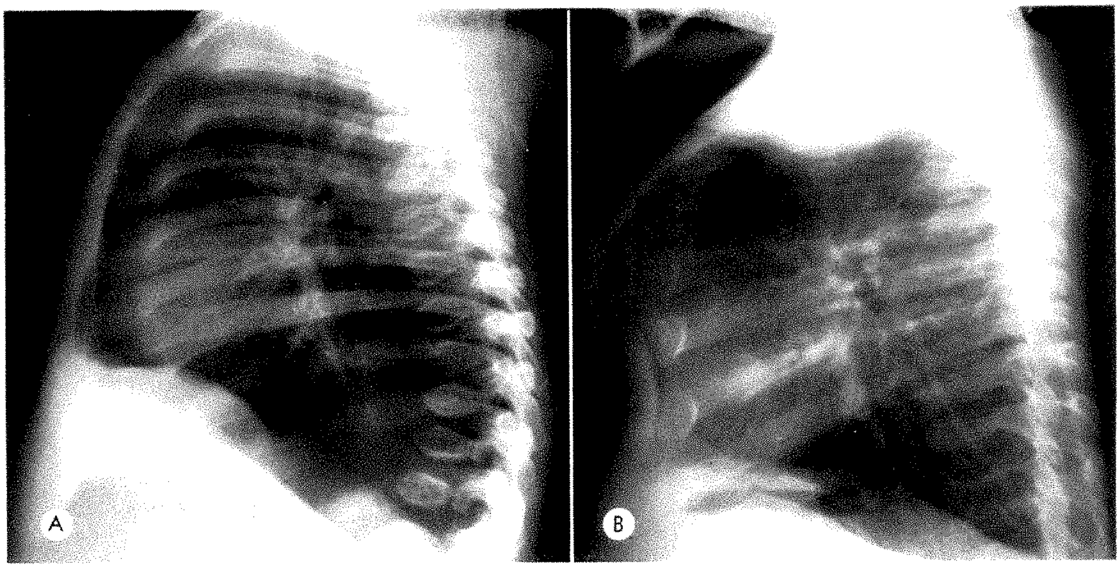

FIG. 9. (*A*) *Young child.* Mild kyphosis and deep thoracic cage. (*B*) *Older child.* Marked sternal protrusion.

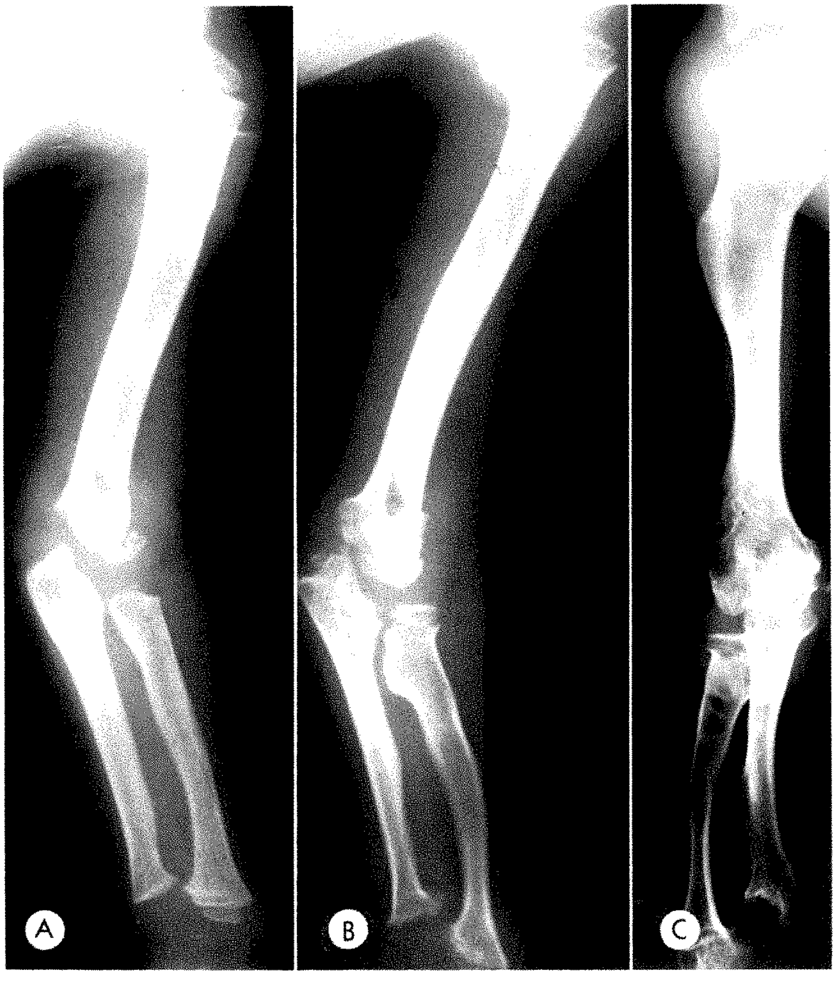

FIG. 10. (*A*) *Young child.* Abnormal modeling of humerus, radius and ulna; mild dorsal angulation of humeral neck. (*B*) *Older child.* Dorsal angulation of humeral neck; widening of proximal radius and ulna. (*C*) *Adult.* Foreshortening of humeral head due to dorsal angulation of neck; prominent deltoid tubercle; widening of humerus, radius and ulna in elbow region. Ulna disproportionately short.

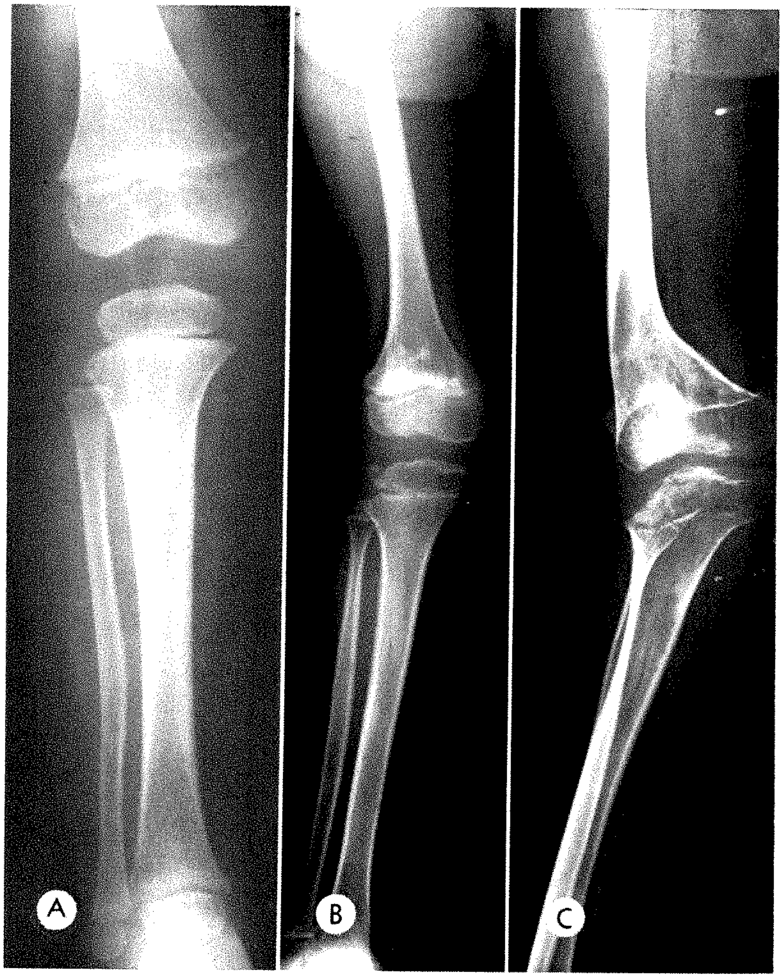

FIG. 11. (*A*) *Young child.* Essentially normal. (*B*) *Older child.* Sclerosis adjacent to distal femoral growth pla and increased trabeculations in distal shaft. Similar changes in proximal tibia; changes at knee contribu ing to genu valgum (see text). (*C*) *Adult.* Similar to older child except for changes due to maturati factors. Diaphyses are normally modeled in distinction to upper extremity.

fusion between the manubrium, body and xiphoid, and there is marked pectus carinatum deformity. The clavicles are relatively normal in shape. The joint space at the shoulder remains wide. The glenoid fossae are shallow. Deformity of the proximal humerus persists. The deltoid tubercle may be prominent. There is relative widening of the ends of the bone in the elbow region and again the proximal ulnar and radial shafts are wide. As mentioned before, the ulna is commonly disproportionately shorter than the radius (Fig. 10*C*).

In the lower extremity, the lower ends of the femur and the upper ends of the tibia are large in relationship to the shafts of tl bones. The same type of change is seen the metaphyseal region as described in la childhood and there is usually deficie ossification of the lateral aspect of proxim tibia. All these changes contribute to tl genu valgum deformity. In the adul diffuse osteoporosis is common, presur ably due to disuse factors (Fig. 11*C*). Tl shafts of the long bones of the lower e tremities are generally quite slender ar without irregularities in their contour distinction to the irregular widening ar narrowing seen in the shafts of the lor bones of the upper extremities (Table IV

TABLE II

DIAGNOSTIC ROENTGENOGRAPHIC FINDINGS AT AGE 2 THROUGH 6 YEARS (IT IS IN THIS PERIOD WHEN THERE IS THE MOST EVOLUTION OF BONE CHANGES)

Case	1	2	3	4	5	6	7	8	9
Teeth Changes	+	+	+	+	NE	+	+	NE	NE
Hypoplastic or Absent Odontoid	+	+	+	+	NE	+	NE	NE	NE
Anterior Tongue Thoracic Bodies	+	+	+	+	+ (min.)	+	+	+	+
Hook-Shaped Lumbar Bodies	+	+	+	+	+	NE	+	+	+
Wide Disk Spaces	+	+	+	+	+	+	+	+	+
Flared Ilia	+	+	+	+	+	NE	+	+	+
Capital Femoral Epiphysis Compression	+ (3½)*	+ (5½)*	+ (5)*	+ (5)*	+ (6)*	NE	+ (6)*	+ (min. at 5)*	±4*
Long Pelvic Configuration	+	+	+	+	+	NE	+	+	+
Coxa Valga	+	+	+	+	+	NE	+	+	+
Deficient Ossification of Superior Acetabula	+	+	+	+	+	NE	+	+	+
Increased Joint of Cleft Hip	+	+	+	+	+	+	+	NE	+ (min.)
Conical Bases of Metacarpals 2–5	+	+	+	+	+ (min.)	NE	+	NE	NE
Normal Constriction of Metacarpals 2–5	+	+	+	+	+	+	+	NE	NE
Carpal Centers	NlN	at 5 yr.	NlN	NlN	NlN	NlN	NlN	NE	NE
Deformity of Distal Radius and Ulna	+	+	+	+	+	+	+	+	NE

+ = present.
DN = decreased number.
NE = not evaluated.
NlN = normal number.
min. = minimal.
± = equivocal.
* age at first examination showing this change.

II. EVALUATION OF CASES IN THE ENGLISH LITERATURE

In Table v an evaluation is presented of the cases considered by Rubin in his textbook, "Dynamic Classification of Bone Dysplasias" (pp. 118–119)[21] as representing spondyloepiphyseal dysplasia congenita (Morquio's disease). This evaluation is based on the roentgenographic, clinical, and biochemical criteria presented above.

III. REVIEW OF CASES IN THE WORLD LITERATURE

Review of the 96 cases in the world literature through 1963, from the recent article

TABLE III

DIAGNOSTIC ROENTGENOGRAPHIC CHANGES AT AGE 6 THROUGH 16 YEARS (IN THIS AGE RANGE THE ESTABLISHED CHANGES PROGRESS IN SEVERITY)

Case	2	3	5	6	7	8	9	10	11	12
Teeth Changes	NE	+	+	+	NE	NE	NE	+	+	+
Hypoplastic or Absent Odontoid	NE	+	NE	+	NE	NE	NE	+	+	NE
Anterior Tongue Thoracic Bodies	+	+	+	+	+	+	+	+	+and flat and rect.	+
Hook-Shaped Lumbar Bodies	+	+	+	+	+	+	+	+	+	+
Wide Disk Spaces	+	+	+	+	+	+	+	+	+ (min.)	+
Flared Ilia	+	+	+	+	+	+	+	+	+	+
Capital Femoral Epiphysis Compression	+ (15)* Neck er.	+	+ (8)* Neck er.	+ Neck er.	+ (11)* Neck er.	+	+ (16)* Neck er.	+	+ (15)* Neck er.	+
Long Pelvic Configuration	+	+	+	+	+	+	+	+	+	+
Coxa Valga	+	+	+	+	+	+	+	+	+	+
Deficient Ossification of Superior Acetabula	+	+	+	+	+	+	+	+	+	+
Increased Joint of Cleft Hip	+	+	+	+	+	+	+ Sub-luxation	+	+	+
Conical Bases of Metacarpals 2–5	+	+	NE	+	+	NE	NE	+	+ (min.)	+
Normal Constriction of Metacarpals 2–5	+	+	NE	+	+	NE	NE	+	+	+
Carpal Centers	DN	DN	NE	DN	DN	NE	NE	DN	small	DN
Deformity of Distal Radius and Ulna	+	+	NE	+	+	NE	NE	+	+	+
Flat or Concave Mandibular Condyle Heads	NE	±	+	+	NE	NE	NE	—	±	NE

+ = present.
NE = not evaluated.
Neck er. = neck erosion.
DN = decreased number.
— = absent.
± = equivocal.
rect. = rectangular.
* age at first examination showing neck erosion.

on Morquio's disease by Maroteaux *et al.*[18] shows that these cases appear to represent true Morquio's disease with the exception of the 4 siblings described by Whiteside and Cholmeley[28] and one of the 2 cases of Shelling.[24] It is probable that Maroteaux *et al.* did not consider this latter case an example of Morquio's disease but no notation to this effect is included in their article.

IV. REVIEW OF CASES SINCE 1963

Review of cases reported in the English language literature as Morquio's disease since the publication of the above 2 sources through 1964, revealed 4 articles on Morquio's disease which included roentgenographic studies. Two of these articles described conditions which were not Morquio's disease.[5,19] Two articles described patients with Morquio's disease but complicated the diagnostic issue by introducing the term Morquio-Ullrich's disease.[8,23]

DISCUSSION

Morquio in 3 articles, 2 in 1929[16,18] and 1 in 1935,[17] described 4 affected siblings in one family. Their normal parents were cousins. He referred to the condition as a familial osseous dystrophy. Brailsford[2] orig-

Table IV

DIAGNOSTIC CHANGES IN THE ADULT

Case	8	9	13	14	15	16
Teeth Changes	+	+	+	+	no teeth	no teeth
Hypoplastic or Absent Odontoid	+	+ hypoplastic	+	+	+ hypoplastic	+
Anterior Tongue Thoracic Bodies	− flat and rect.	+	+ and flat and rect.	+ and flat and rect.	+ and flat and rect.	− flat and rect.
Hook-Shaped Lumbar Bodies	+	+	+	+	+	+
Wide Disk Spaces	±	+	+	+	+	+
Flared Ilia	+	+	+	+	+	+
Absent Femoral Head Eroded Femoral Neck	+	+	+	+	+	+
Long Pelvic Configuration	+	+	+	+	+	+
Coxa Valga	+	Neck eroded, head absent	± Neck short, completely eroded	± Neck short, completely eroded	± Neck short, completely eroded	± Neck short, completely eroded
Deficient Ossification of Superior Acetabula	+	+	+	+	+	+
Increased Joint Cleft of Hip	− subluxation	− subluxation	+	+	+	− subluxation
Conical Bases of Metacarpals 2–5	−	−	+ mild	+ mild	−	+ mild
Normal Constriction of Metacarpals 2–5	+	+	+	+	+	+
Carpal Centers	DN	DN	DN	DN	DN	DN
Deformity of Distal Radius and Ulna	+	+	+	+	+	+
Flat or Concave Mandibular Condyle heads	+	+	+	+	+	+

+ = present.
− = absent.
± = equivocal.
DN = decreased number.
rect. = rectangular.

TABLE V

EVALUATION OF CASES CONSIDERED BY RUBIN TO REPRESENT SPONDYLOEPIPHYSEAL DYSPLASIA CONGENITA (MORQUIO'S DISEASE) ON BASIS OF CURRENT ROENTGENOGRAPHIC, CLINICAL AND BIOCHEMICAL CRITERIA

Author	Year	Age and Sex	Journal	Comment—Primarily Based o Roentgenographic Findings
Morquio	1929	female, 14; male, 8	*Bull. Soc. pédiat.*, *27*, 145	Original cases of Morquio
Morquio	1929	female, 14; male, 8	*Arch. méd. d'enf.*, *32*, 129	Original cases of Morquio
Brailsford	1929	male, 4	*Am. J. Surg.*, *7*, 404	Original case of Brailsford
Brailsford	1931	male, 4	*Brit. J. Radiol.*, *4*, 83	Original case of Brailsford
Dale	1931	female, 25; male, 11 female, 4 (in 2 families)	*Acta radiol.*, *12*, 337	Morquio's disease
Ruggles	1931	8 children (5 families)	AM. J. ROENTGENOL. & RAD. THERAPY, 25, 91	Illustrated children have Morq disease
Wright	1931	male, 10	*Proc. Roy. Soc. Med.*, *24*, 283	Not Morquio's disease on basi pelvic and hand configuration
Meyer and Brennemann	1932	male, 10 mo.–6½ yr.	*Am. J. Dis. Child.*, *43*, 123	Not Morquio's disease on basi pelvic configuration with vara; dwarfed in 1st year of
Turner	1933	male, 10	*Proc. Roy. Soc. Med.*, *26*, 775	Not Morquio's disease on basi pelvic and spine configuration
Brown	1934	female, 9; female, 12 female, 38	*M. J. Australia*, *1*, 598	Not Morquio's disease on basi pelvic and spine configuratio another article on these patie
Warkany and Mitchell	1934	male, 12	*J. Pediat.*, *4*, 734	Not Morquio's disease; agree Rubin; this is a type of mul epiphyseal dysplasia
Neale and Hucknall	1934	male twins, 6	*Arch. Dis. Child.*, *9*, 51	Morquio's disease
Morquio	1935	male, 15; male, 19	*Arch. méd. d'enf.*, *38*, 5	Morquio's disease, siblings of ori cases
Brown and Summerfeldt	1936	male, 9; male, 11	*Arch. Dis. Child.*, *11*, 221	Morquio's disease
Hirsch	1937	female, 51; male, 19 female, 17 male twins, 12 male, 10; male, 5	*J. Bone & Joint Surg.*, *19*, 297	Cases 6 and 7, Morquio's dis Cases 1–5 not Morquio's dis they represent a type of mul epiphyseal dysplasia
Pohl	1939	female, 22	*J. Bone & Joint Surg.*, *21*, 287	Not Morquio's disease on basi spine configuration and also s

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* Brown and MacDonald. *Australian & New Zealand J. Surg.*, 1933, *3*, 78.

TABLE V (*Continued*)

Author	Year	Age and Sex	Journal	Comment—Primarily Based on Roentgenographic Findings
				and pelvis configuration in infancy in another article†
Crawford	1939	male, 9	*Arch. Dis. Child.*, *14*, 70	Not Morquio's disease on basis of spine, pelvic and hand configuration
Reeves and Baylin	1941	male, 5; female, 6	*Radiology*, *36*, 363	Not Morquio's disease on basis of pelvic and hand configuration
Einhorn *et al.*	1941	male, 12; male, 7 male, 7	*Am. J. Dis. Child.*, *61*, 776	Cases 1 and 2, Morquio's disease (roentgenograms of Case 2 are labeled Case 3 in article and vice versa); Case 3 not Morquio's disease on basis of spine configuration
Fanell *et al.*	1942	female, 19; male, 14	*Arch. Neurol. & Psychiat.*, *48*, 456	Not Morquio's disease on basis of hand configuration and skull (teeth) in another publication on same patients‡
Russo	1943	female, 26	*Radiology*, *41*, 42	Negro adult with Morquio's disease
Einhorn *et al.*	1946	male, 10	*Am. J. Dis. Child.*, *72*, 536	Case 2 of 1941 article by Einhorn *et al.*; cord damage at autopsy
Scott and Rotondo	1946	female, 5	*Arch. Pediat.*, *63*, 261	Negro child with Morquio's disease
Fairbank	1949		*J. Bone & Joint Surg.*, *31B*, 291	2 patients have Morquio's disease; 2 have different conditions on basis of spine and pelvic configuration
Jackson	1951	female, 52; male, 56 female, 62	*J. Bone & Joint Surg.*, *33B*, 420	These cases have been referred to by some recent authors as diastrophic dwarfism; personally favor form of multiple epiphyseal dysplasia with club feet
Whiteside and Cholmeley	1952	male, 1–11 yr. male, 2–9 yr. female, 6–12 mo. male, 2 mo.–2½ yr.	*Arch. Dis. Child.*, *27*, 487	Not Morquio's disease on basis of pelvic configuration at 11 years
Brailsford	1952	male, 14–18 yr. female, 3½–24 yr. male, 10 female, 22	*J. Bone & Joint Surg.*, *34B*, 53	Cases 2, 4 and 5, Morquio's disease; Case 3 not Morquio's disease on basis of pelvic configuration
Toma	1953	female, 7 female, 17	*S. Clin. North America*, *336*, 1765	Case 1 has Morquio's disease; Cases 2 and 3 not Morquio's disease on

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† Wheeldon. *Am. J. Dis. Child.*, 1920, *1*, 19.

‡ Benda. Developmental Diseases of Mentation & Cerebral Palsies. Grune & Stratton, N.Y., 1952, p. 434.

Table V (*Continued*)

Author	Year	Age and Sex	Journal	Comment—Primarily Based on Roentgenographic Findings
		female, 4		basis of pelvic and hand configuration
Lipshutz	1955	male, 3 female, 7	*J. Pediat.*, *46*, 403	Not Morquio's disease on basis of hand and pelvic configuration
Melkis and Mautner	1958	male, 28	*Arch. Pediat.*, *75*, 285	Morquio's disease
Hobaek	1961	families 7–27 except for 19 (according to Rubin)	Problems of Hereditary Chondrodysplasia, Oslo Univ. Press, Oslo 1961	Only families 7–10 have individuals with Morquio's disease
Schenk and Haggerty	1964	male, 12; female, 17	*Pediatrics*, *34*, 839	Morquio's disease

inally described a 4 year old patient under the term chondro-osteo-dystrophy and gave a detailed description of the roentgenographic findings.

Morquio in his article in 1935 mentioned 8 articles by other authors between 1929 and 1935 describing cases considered by these authors to have Morquio's disease. He rejected the cases in 2 of these articles.[23,27] Of the remaining 6 articles,[1,3,6,15,22,26] 3 reported true Morquio's disease patients.[1,22,26]

Brailsford included a large number of cases under his term chondro-osteo-dystrophy. He divided them into the following 4 groups, based on severity and regional distribution of deformity as seen on roentgenograms: Group A, severe generalized cases, in which he placed his first patient; Group B, where the active progression of the disease appeared to cease before puberty; Group C, where the changes were confined chiefly to the spine, hips and knees. Interestingly enough, he included Morquio's patients in this group; and Group D, where changes were confined to the vertebral column. His classification included a number of different conditions in each group and provided a precedent for including any condition presenting a short spine as Morquio's disease.

It is our opinion that the first case published under the name of Morquio's disease in the American literature, by Meyer and Brennemann,[15] represented another condition. Roentgenograms and photographs of a 5 year old boy with the same condition as Meyer and Brennemann's patient are used to illustrate the section on Morquio's disease in Caffey's textbook, "Pediatric X-Ray Diagnosis."[4] We have examined patients with this condition and found that they do not have corneal opacities, dental abnormalities or keratosulfate in the urine. They are dwarfed at birth unlike true Morquio's disease patients, and there is no evidence to our knowledge that this condition has an autosomal recessive mode of genetic transmission as is true in Morquio's disease.

If we accept Rubin's suggested distinction between bone dysplasia and bone dystrophy, Morquio's disease must be considered a bone dystrophy rather than a bone dysplasia as Rubin classified it. Nosologically, Morquio's disease belongs with the other mucopolysaccharidoses of which the Hurler syndrome or gargoylism is the best known example.

We believe that spondyloepiphyseal dysplasia congenita, Rubin's term for Morquio's disease, is not a suitable descriptive name since the changes are not limited to the spine and epiphyses. It also implies a relationship which probably does not exist between Morquio's disease and the other conditions to which the name spondyloepiphyseal dysplasia has been applied. In our opinion, of the 80 childhood cases in Rubin's review of the literature, 41 represented Morquio's disease and 39, some other condition. This is excluding the children with spondyloepiphyseal dysplasia of the pseudoachondroplastic type and spondyloepiphyseal dysplasia tarda. We have examined patients with both the pseudoachondroplastic type and several conditions which might be termed spondyloepiphyseal dysplasia tarda, including the X-linked condition originally described under this name by Maroteaux, Lamy and Bernard[13] as well as conditions with a dominant mode of genetic transmission. No corneal opacities, dental abnormalities or abnormal urinary mucopolysaccharides were present in these patients. There is no evidence in our opinion to suggest that the term spondyloepiphyseal dysplasia as applied to these conditions is anything other than descriptive of apparent regional involvement. The evolution of changes in the individuals with the pseudoachondroplastic type suggest that this condition may be closely related to the multiple epiphyseal dysplasia group. While the articular cartilage does undergo early degeneration in the X-linked condition described under this name by the French authors, the fibrocartilage of the intervertebral disks also shows early pathologic change. This condition has a distinctive roentgenographic appearance in the lumbar spine of the adult[11] and no evidence is currently available to suggest that it has the same etiology as the dominant condition illustrated by Rubin under this name in his text. Certainly, the roentgenographic picture is different. While most patients with Morquio's disease have been correctly diagnosed in the past, a number of other apparently unrelated conditions have likewise been diagnosed as Morquio's disease.

Recently the term Morquio-Ullrich's disease has been introduced into the English literature. Zellweger *et al.*[29] described Morquio-Ullrich's disease as a condition in which, in addition to the bone changes of Morquio-Brailsford's disease, there are corneal opacities and dental abnormalities. Riley granulations in the white blood cells, deafness, and hepatomegaly were present in some cases. These authors found evidence of abnormal mucopolysaccharide excretion in the urine of their 2 patients, and in the 1 patient tested the results suggested that this probably represented keratosulfate. They contrasted the condition with Morquio-Brailsford's disease where no abnormalities outside of the musculoskeletal system were described by the original authors.

The present series and that of Maroteaux *et al.*[14] are the only large series in which corneal opacities, dental abnormalities and abnormal urinary mucopolysaccharide have been specifically looked for. Maroteaux *et al.* examined 8 children with the bone changes described by Morquio and Brailsford and found corneal opacities and dental abnormalities in all. In the 6 children tested, keratosulfate was demonstrated in the urine. They stated that other patients with platyspondyly did not show these findings. Our personally examined series of 10 patients with Morquio's disease showed the same findings as those of Maroteaux *et al.* This was also true of the bone and teeth changes in the roentgenographic studies in which teeth changes could be evaluated in 6 additional Morquio disease patients. The 2 brothers described by Zellweger *et al.*, in our opinion, have the condition originally described by Brailsford and Morquio. The same clinical, roentgenologic and biochemical changes were described in 2 siblings by Robins *et al.*[20]

That Morquio and Brailsford did not comment on the corneal opacities is not surprising since these are frequently only visible on slit lamp examination. Brailsford specifically commented on the numerous dental caries in his 4 year old patient. The lateral skull roentgenograms in Brailsford's 1931 article and in Morquio's 1935 article strongly suggest the type of dental changes seen in our patients. Present evidence indicates that Morquio-Brailsford's disease is a mucopolysaccharidosis, and that Morquio-Ullrich's disease is a superfluous term. As one of the authors (LOL) has previously suggested,[12] we prefer the designation, KS mucopolysaccharidosis of Morquio. This combines the biochemical abnormality with the time honored eponym for this condition.

Maroteaux *et al.* reviewed the world literature and found 96 published cases which they accepted as Morquio's disease. They seem to have excluded one of Shelling's 2 cases although they do not specifically state so in their bibliography. Certainly one of Shelling's cases does not represent Morquio's disease since the clinical course and bone changes are different in this case, and we have found no corneal opacities, dental abnormalities or abnormal mucopolysaccharide excretion in our personal cases with this condition. Maroteaux *et al.* include the 4 siblings reported by Whiteside and Cholmeley as examples of Morquio's disease. The bone changes in these children are clearly different from those in Morquio's disease. This is most convincingly demonstrated on the roentgenogram of the pelvis of a patient at the age of 11 years in Whiteside and Cholmeley's article. We have found no corneal opacities, teeth changes or abnormal mucopolysaccharide in a patient with similar bone changes. The article of Maroteaux *et al.*[14] with its extensive bibliography is recommended to the interested reader.

Disability due to bone deformity at the hips and knees (genu valgum) was recognized in Morquio's disease. Some individuals have pulmonary complications due to chest deformity, and murmurs in the aortic region have been described. Hearing defect is common in individuals with this condition. Five of the 6 adults in our series had pathologic long tract signs referable to the high cervical region. It seems likely that spinal cord damage presumably due to a combination of bone deformity and ligamentous relaxation may prove to be a common serious medical complication in the affected older child and adult. Einhorn *et al.*[7] and Hobaek[9] reported similar neurologic complications. A detailed discussion of clinical features and biochemical changes is outside of the scope of this article.

We believe it is important to emphasize that subsequent children of parents with an affected child may be similarly affected since the condition has an autosomal recessive mode of genetic transmission. The parents should be informed of this possibility. Since affected children appear completely normal or only mildly deformed for several years, the eventual marked dwarfism and deformity are perhaps more difficult for both the parents and child to accept than if the deformity were present from birth. Affected children should be evaluated neurologically at regular intervals and parents informed that cervical spine surgery may eventually be necessary to prevent the serious cord complications which were common in our older patients.

The discussion of the roentgenographic differential diagnosis is difficult because so many conditions have been and are still called Morquio's disease that clearly do not represent the condition originally described by Morquio and Brailsford. In our opinion, present evidence indicates that patients who do not show the roentgenographic features discussed in the body of this article do not have Morquio's disease. Robins *et al.*[20] have described a partial roentgenographic examination in an 8 month old infant who was clinically normal. This child was examined because of an older affected sister. The studies showed the diagnostic changes outlined in the section on the roentgenographic findings in early childhood.

Some authors have considered it impossible to differentiate roentgenologically between Morquio's disease and the conditions of the Hurler syndrome which are also mucopolysaccharidoses. This has not been our experience in 50 chemically or histologically confirmed cases. Although the hook or beak-shaped configuration of the vertebral bodies in the thoracolumbar junction and upper lumbar spine may be identical in Morquio's disease and in the Hurler syndrome, we have never seen flattened vertebrae throughout the entire thoracic region in cases with the Hurler syndrome. Generally, it is the severely affected individuals with Hurler syndrome who show gross bone changes which can be confused with those of Morquio's disease. These individuals' metacarpal shafts show a lack of normal constriction and usually marked expansion of the metacarpal shafts. This does not occur in Morquio's disease in our experience. The pelvis and hip configuration in severely affected Hurler syndrome patients is also different from that seen in Morquio's disease.

Two more general points in the differential diagnosis should probably be emphasized. Babies who are dwarfed at birth and during the first year of life do not have Morquio's disease, and coxa vara is never seen in Morquio's disease.

Our experience would indicate that the roentgenographic diagnosis of Morquio's disease can be made by the age when signs and symptoms due to the condition cause parents to bring the child to the physician, that is, around 1 year of age. This is before diagnostic clinical deformities develop.

A roentgenographic diagnosis of Morquio's disease is the most practical mode of early diagnosis because, while equally specific, the biochemical tests for keratosulfate are relatively difficult to perform, time consuming, and available in only a few medical centers at the present time.

SUMMARY

The criteria for the roentgenologic diagnosis of Morquio's disease are presented, based on an analysis of 16 patients.* Diagnostic findings are seen in the teeth, spine, pelvis and the hand and wrist. The diagnosis can be established at an age when signs and symptoms become evident.

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* Since this article was submitted 11 additional cases of Morquio-Brailsford's disease have been reviewed. The clinical and roentgenographic features in these cases were identical to those of the 16 cases analyzed in this article.

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VOL. 97, NO. 1

COMBINED METAPHYSEAL AND EPIPHYSEAL DYSOSTOSIS*

REPORT OF TWO CASES—ONE, IN WHICH METAPHYSEAL CHANGES PREDOMINATE, AND A SECOND ONE, IN WHICH EPIPHYSEAL CHANGES ARE MORE MARKED

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DEFECTIVE metaphyseal ossification without known biochemical changes—the metaphyseal dysostosis syndrome—is a common familial disease with a roentgenographic appearance which has many variations. Sometimes, the disease is well defined and consistent,[5,6,8,10] but many times it does not fit any recognized type and is difficult to classify. Catal[3] recognized these inconsistencies several years ago, and pointed out that occasionally the metaphyseal and epiphyseal types of dysostosis overlap. These types were called dysostosis meta-epiphysealis or epi-metaphysealis, depending on predominant changes. These are purely general descriptive terms founded on roentgenographic changes. They are convenient for clinical use but they do not identify the causal mechanisms.

Both Ribbing and Fairbank have described disturbed epiphyseal ossification, not associated with biochemical changes in the urine or blood, in which the metaphyses are unaffected. The process is now called multiple epiphyseal dysplasia—a common familial disease. There are many variants in the distribution and the degree of these epiphyseal lesions. In this paper, 2 examples of combined metaphyseal and epiphyseal lesions in single patients are reported. In the first patient, the metaphyseal changes predominated (meta-epiphyseal dysostosis) and in the second, the epiphyseal changes were more marked (epi-metaphyseal dysostosis).

REPORT OF CASES

CASE I. *Meta-epiphyseal dysostosis.* S.R., a boy, was the product of a 6th pregnancy. At 7 years of age, he was examined at another hospital. No known members of his family had had bone disease. At birth, he weighed 4,000 gm. and was considered normal. He was breast fed during the first 6 months of life. During the third week of age, the sternum was noted to be prominent and the knees and ankles were thickened. These changes increased and the diagnosis of "pigeon breast" deformity was made at 6 months of age. After a bout of pneumonia in the 3rd month of life, he was noticed to be shorter than normal. The deficiency in stature became more conspicuous with advancing age. His motor functions were retarded. He first raised his head at 5 months, first sat at 14 months, stood at 21 months and walked at 25 months. His gait was always stiff. His steps were short and he fatigued easily.

At the time of this examination at the age of 7 years, his height was 82 cm. (normal 120 cm.) and weighed 11 kg. (normal 22 kg.). The head was normal. Marked shortening of the trunk with kyphoscoliosis of the lower thoracic and upper lumbar spine and lordosis of the lower lumbar spine were present. There was a "pigeon" deformity of the chest; the body of the sternum protruded more than 2 cm. forward in relation to the manubrium. A Harrison's groove and costal rosary were present. The proximal parts of the upper and lower extremities were shortened but the hands and feet were normal in length. The femora were bowed laterally and anteriorly. Both feet were flat. In the organs of the chest and the abdomen, no abnormalities were found. The mobility in the large joints of the extremities was limited. Mental development was normal. Laboratory examinations showed normal values of calcium, phosphorus and alkaline phosphatase. A routine examination of the urine and blood was also normal.

Roentgenologic study disclosed changes sug-

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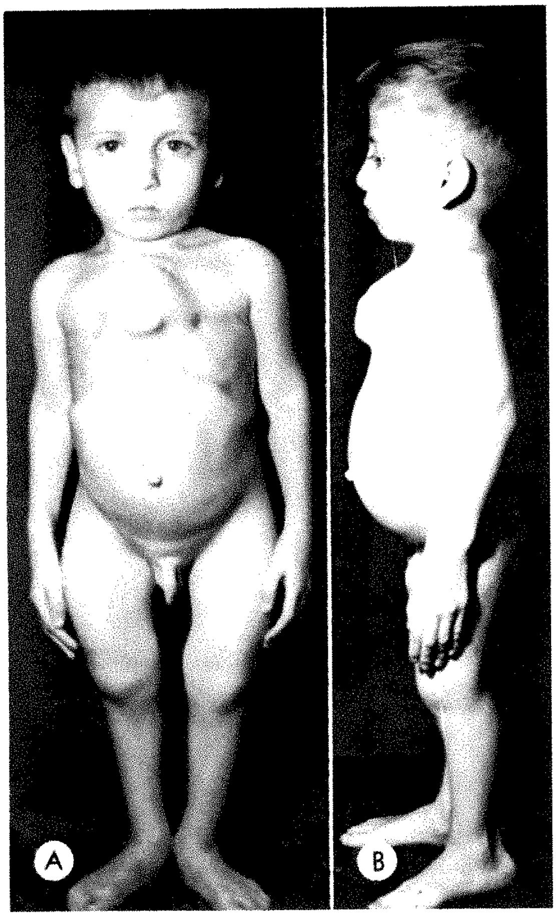


FIG. 1. Case 1. Photographs of S.R. at 8 years, 9 months of age. (*A*) Frontal and (*B*) lateral views. Height is 35 per cent less than the mean value for Polish boys of 9 years. The head is normal. The shortening of the extremities is of the root type. The hands are relatively elongated. The thighs are bowed anteriorly and laterally. Both feet are flat and everted. The trunk is also shortened. The costal arches reach almost to iliac crests. The deformed sternum and anterior parts of the ribs project forward to form a "pigeon" chest; Harrison's grooves and a costal rosary are also visible. Kyphoscoliosis of the lower thoracic and upper lumbar spine, and lordosis in lower lumbar levels were visible in the physical examination. The sacrum was rotated upward and backward to a near horizontal position. Mental development was normal.

gestive of dysostosis enchondralis. On the basis of all the findings, the diagnosis of Morquio-Brailsford's disease was finally made.

The child was re-examined on January 4, 1965, at the age of 8 years, 9 months in the First Pediatric Clinic of the Medical School in Poznan, Poland. His morphologic status in comparison with the findings in the other hospital had not changed (Fig. 1, *A* and *B*). The child had grown 3 cm. during 20 months and weighed 13 kg. His measurements are shown in Table I.

As the history and the physical findings were not characteristic for Morquio-Brailsford's disease, further hematologic, biochemical, ophthalmologic and roentgenologic studies were performed. The hematologic examination did not disclose Adler granulations, Buchot cells, Lorincz-Pearson granulations or Gasser cells. The level of mucopolysaccharides in urine, measured by urine chromatography, was normal. No corneal opacities were seen in slit-lamp examinations. The significant roentgenographic changes were confined to the metaphyses and epiphyses of the tubular bones, with predominance of the process in the metaphyses (Fig. 2, 3, 4, 5 and 6). The ends of the shafts of all the tubular bones—the elbow region excluded—were widened and irregularly mineralized in a deep terminal zone of irregular density, extending laterally and medially beyond the ends of the epiphyseal ossification centers. These changes were best seen at the proximal ends of the femoral and humeral shafts and the distal ends of the tibial, radial and ulnar bones. The transverse clear zones between the ends of the shafts and the epiphyseal ossification centers were deepened. The epiphyseal ossification centers were hypoplastic, flattened and of irregular structure. The degree of the involvement of the epiphyseal ossification centers corresponded to the changes in the metaphyses. The calvarium (not illustrated) was thin with

TABLE I

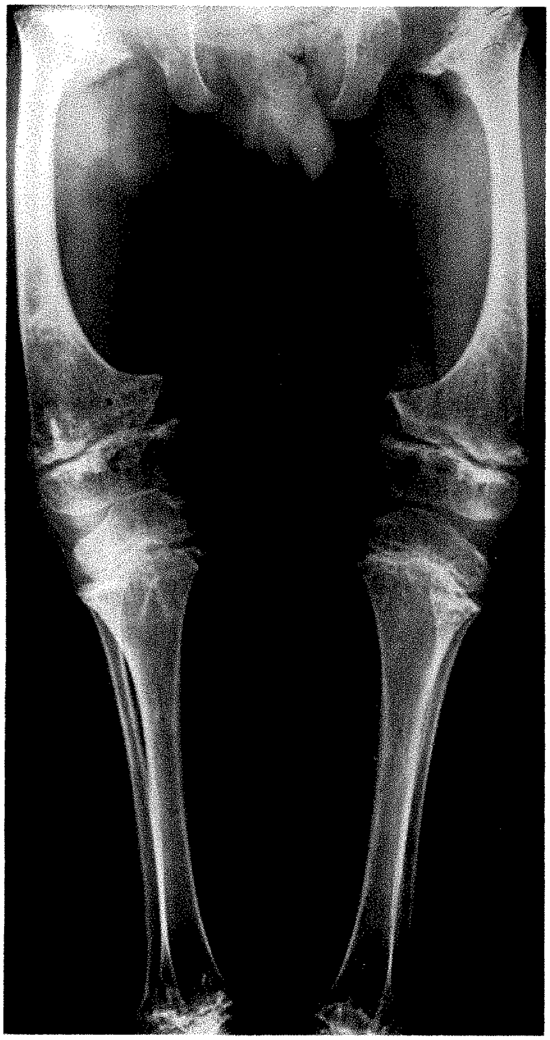
CASE I

Height	85 cm.
Circumference of head	51 cm.
Circumference of chest (at the level of protruding corpus sterni)	55 cm.
Span	106 cm.
Upper limb (acromion to third finger)	15 cm.
Forearm and hand (radial point to third finger)	27 cm.
Hand (styloid point to third finger)	14 cm.
Lower extremities (anterior superior iliac spine to floor)	42 cm.
Leg (tibial point to floor)	23 cm.
Foot	18 cm.
Weight	13 kg.

retarded differentiation of the inner and outer tables and the diploic space. The pituitary fossa was normal. There was no evidence of dysostosis in the cartilaginous bones of the base of the skull. The teeth were crowded but otherwise the dental development was normal. The skeleton was generally rarefied.

Case II. *Epi-metaphyseal dysostosis.* D.H., a girl 39 months of age, born on January 15, 1953, came to the First Pediatric Clinic in Wroclaw, Poland, because, on roentgenographic examination, changes suggestive of rickets or of an endocrine disease had been found.

She was the product of a first pregnancy. The parents were young and healthy. A brother and other relatives were normal in stature. There are no known bone diseases in the family. Gesta-



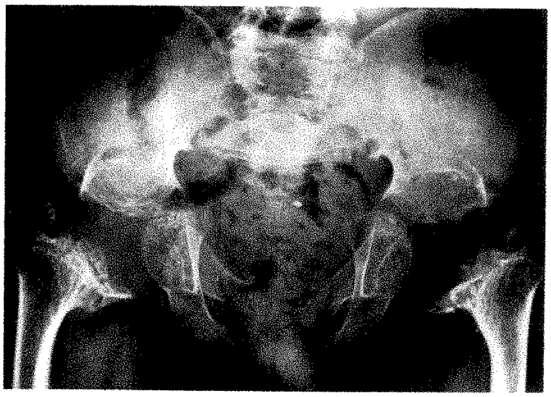


Fig. 3. Case I. The pelvis and hips in frontal projection. The necks and heads of the femora are not calcified and the proximal ends of the shafts are cupped and flare widely. Small irregular ossification centers are visible laterally above the irregular metaphyses. Coxa vara is marked on both sides. The proximal femoral metaphyses are widened and irregularly ossified in a deep zone of irregular density. Long spurs project medially off the medial ends of the femoral metaphyses. The submetaphyseal spongiosa is deformed and irregular both in pattern and density. The convex rims of the deepened acetabular cavities are irregular. The cartilaginous parts of the pelvis are widened. The iliac bones are shortened in the longitudinal diameter, which is reflected in the longitudinal shortenings of the sciatic notches. The sacrum articulates low on the ilia and is tilted backward and upward.

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Fig. 2. Case I. Changes in the lower extremities. Both femora are bowed laterally. The transverse clear zones between the ends of the shafts and the epiphyseal ossification centers are deepened. The femoral metaphyses are widened and irregularly ossified in a deep zone of about 0.5 to 1.5 cm. of irregular density which extends laterad and mediad beyond the ends of the epiphyseal ossification centers. There are some sharply defined radiolucent patches in some of the irregularly ossified metaphyses. The proximal ends of the tibial shafts and the distal ends of the femoral shafts are convex; those of the fibulae are straight. The submetaphyseal spongiosa is deformed and irregular both in pattern and density. Deeper in the shafts, the spongiosa has a ground glass appearance. The mineralization, as well as the thickness of the cortical walls, is decreased. The bases of the ossification centers show marginal dysostosis in zones, several millimeters deep, of irregular ossification. These changes are most marked in the distal tibial epiphyses, which in their middle and outer two-thirds, are flattened and irregularly mineralized. The distal fibular epiphyses are normal.

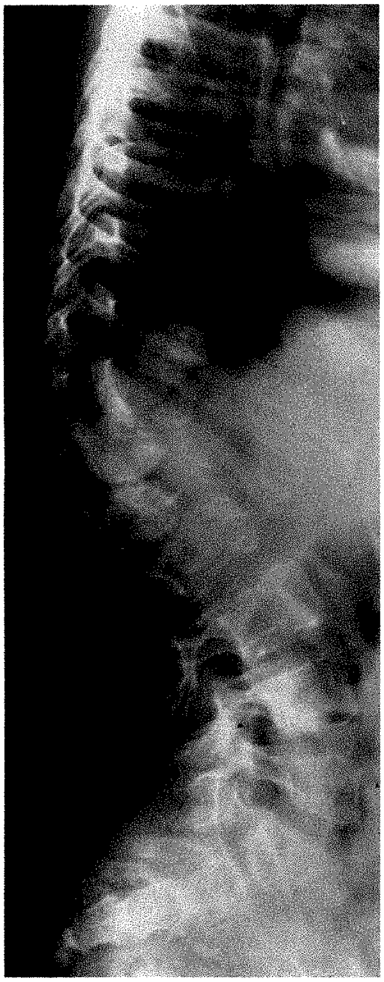

Fig. 4. Case i. The vertebral column in lateral projection. The lower thoracic segments are kyphotic and the lower lumbar are scoliotic. The vertebral bodies are flattened most in the thoracic segments above the kyphosis. Both the upper and lower edges of vertebral bodies are uneven. The intervertebral spaces from the 10th thoracic to the 1st lumbar are narrowed. The vertebral bodies of the 12th thoracic and 1st lumbar are hypoplastic and irregular in structure and density. They are displaced backward, upward and to the left. In frontal projection, not shown here, the right ribs from 4 to 7 were hypoplastic in their posterior parts and tilted downward.

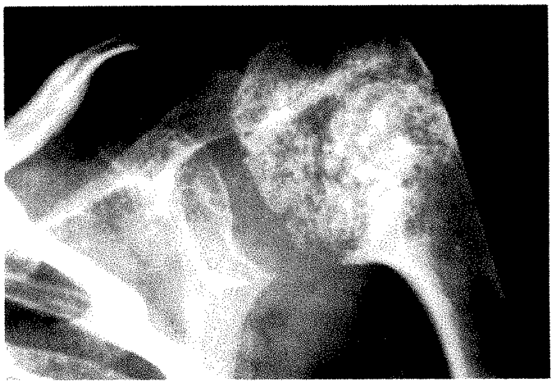

Fig. 5. Case i. Both humeri were shortened. The provisional zones of calcification are not well seen because they are superimposed on the humeral heads. The proximal ends of the humeral shafts and its ossification center are widened and both are irregularly ossified. This irregular metaphyseal zone is obliquely placed on the shaft with the medial end distalward. The submetaphyseal spongiosa is a deformed and irregular mixture of spotty sclerosis and rarefaction. The same pattern of the spongiosa was present, although mild in degree, at the distal end of the humerus, not shown here. The cortical walls of the humeral bones are thin. The metaphysis and the ossification centers were fragmented medially. The glenoid cavity of the scapula is shallow and its rim is irregular. The vertebral bodies in the cervicothoracic levels, in roentgenograms not shown here, were flattened and rarefied.

tion and delivery were normal. This apparently normal, newborn infant weighed 2,900 gm. Her length was not known. She was breast fed during the first 6 months. She received vitamin D after the 12th month. Structural abnormalities were not observed during the first year although her motor development was slightly retarded. She sat at the age of 8 months, stood at 9 months, and walked at 13 months. Her first tooth appeared during the twelfth month.

When she started to walk, her gait was stiff with small steps, and she shifted rather than walked. At that time, rickets was suspected and the child received massive doses of vitamin D.

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Fig. 7. Case ii. D.H., at 12 years of age. (*A*) Frontal, (*B*) lateral and (*C*) dorsal views. The trunk is shortened in relation to arms and legs. A high sternal bulge and costal knobs are visible in the shortened thorax. The spine is bent dorsally and laterally. Both knees deviate medially. Height was $\frac{4}{5}$ of average for Polish girls 12 years of age. Thighs are widely spaced and are shortened. Upper arms are shortened due to deformities at the proximal ends of the humeri. Upper maxilla is large and the lower maxilla also appears to be larger than normal.

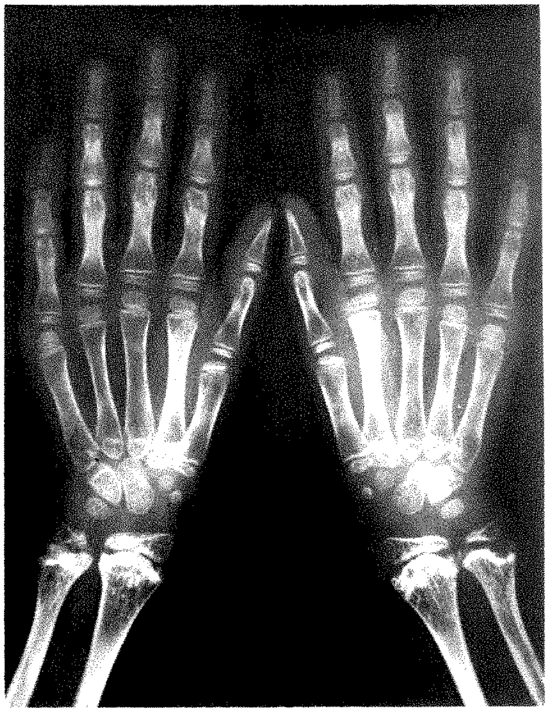

Fig. 6. Case I. Hands and wrists in frontal projection. The metaphyses of the radii and ulnae are irregularly mineralized in contrast to the relatively smooth edges of their epiphyseal ossification centers. The round bones in the wrists are normal. At the distal ends of the first metacarpals and the proximal ends of metacarpals 2, 3, 4 and 5, there are independent and partially fused pointed accessory ossification centers. The epiphyseal ossification centers in metacarpals 1, 2 and 3, especially 1 and 2, are flattened longitudinally. Maturation is normal.

In spite of this treatment and constant medical supervision, her gait worsened, and, at the age of about 2 years, she became knock-kneed. She walked reluctantly and fatigued easily.

At 3 years of age, her height was 81 cm. (normal 95 cm.); and her weight was 11 kg. (normal 15 kg.). The physical examination disclosed a normal head, Harrison's grooves, costal rosary, lumbar lordosis and knock-knees. The

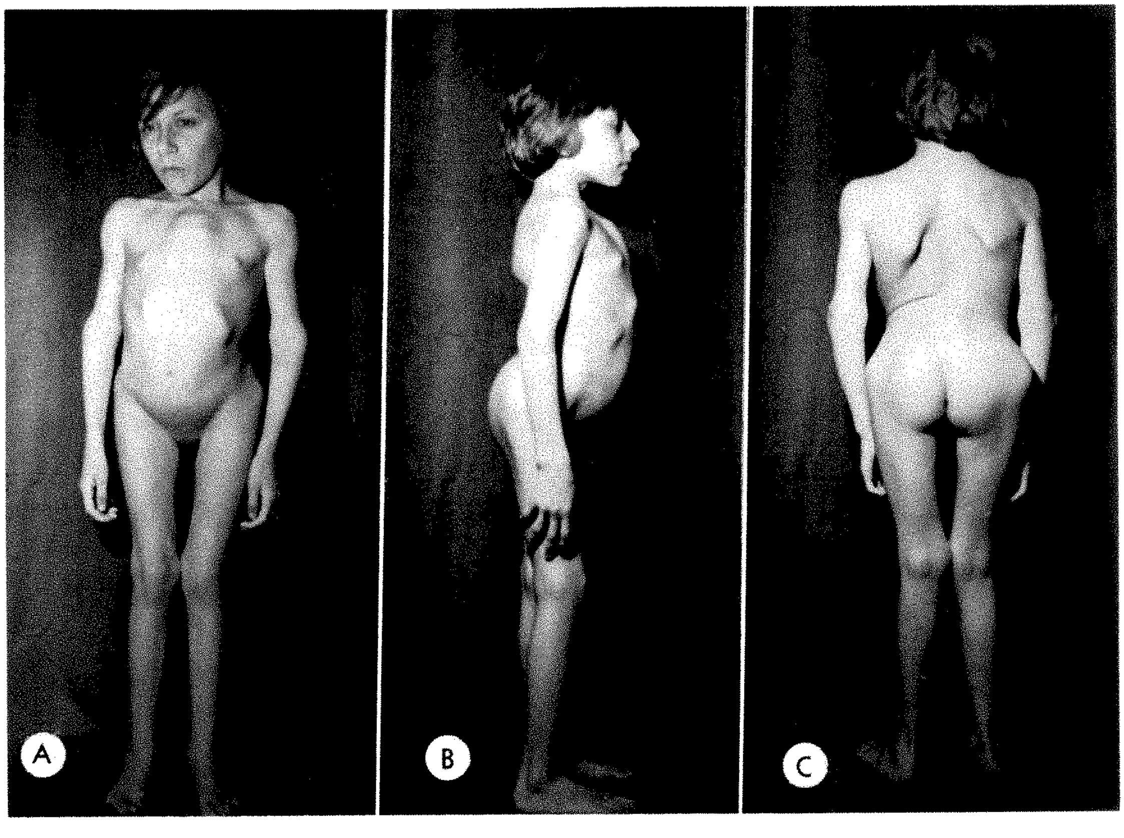

Table II

case ii

Height	115 cm.
Circumference of head	52 cm.
Circumference of chest	65 cm.
Span (arms)	124 cm.
Upper limb (acromion to third finger)	53 cm.
Forearm and hand (radial point to third finger)	32 cm.
Hand (styloid point to third finger)	15 cm.
Lower extremities (anterior superior iliac spine to floor)	65 cm.
Leg (tibial point to floor)	29 cm.
Foot (malleolus to floor)	3 cm.
Weight	20 kg.

gait was abnormal with shifting, short steps. Her mental development was normal for her age. Roentgen examination showed that the femoral heads, humeral capitella, and proximal fibular and distal radial epiphyses were not ossified. All other epiphyseal ossification centers were small for her age and somewhat flattened. Those at the knees and distal tibial, distal fibular and proximal humeral epiphyses showed, in addition to the hypoplastic changes, an uneven, porous density. The ends of the shafts of the long bones were widened and irregular. These changes were more marked in the lower extremities. The carpal ossification centers were small. Maturation of bones in hands and wrists according to the standards of Greulich and Pyle equalled 3 years. The findings in routine examinations of the blood and urine as well as the level of serum calcium were all normal.

She was re-examined again at the age of 12 years in the First Pediatric Clinic in Poznan. Her height then was 115 cm. (normal 150 cm.) and weight 20 kg. (normal 41 kg.). A disproportioned dwarfism, with marked shortening of the trunk and the extremities was present (Fig. 7, *A*, *B* and *C*). The thoracic spine was kyphoscoliotic and the lumbar spine was scoliolordotic. The chest showed a pigeon-breast deformity with Harrison's groove and costal rosary. The metaphyseal zones of the long bones were widened. Muscular development was poor. The active as well as passive movements at the large joints were limited. She walked with difficulty, using short steps and, after walking a short distance, she became tired. Her mental development was excellent and she was the best pupil in her class at school. Corneal opacities were not seen in the slit-lamp examination. The routine examinations of the blood and urine as well as the values of calcium, phosphorus and alkaline phosphatase in the serum, urine chromatography, and the level of urine mucopolysaccharides were normal. Her measurements are shown in Table ii.

The roentgenographic examination showed a generalized osteoporosis of the whole skeleton. The diagnostic roentgenographic changes (Fig. 7 through 11) were confined to the epiphyses

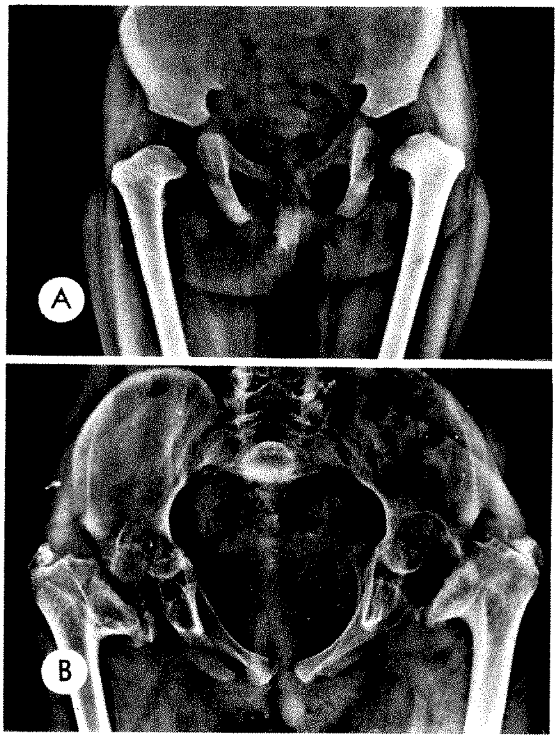

Fig. 8. Case ii. (*A*) Pelvis at 3 years of age and (*B*) at 12 years of age. At 3 years the femoral necks are short and the femoral ossification centers have not yet appeared. The ischiopubic synchondroses are widely open. All of the bones are too small. At 12 years severe varus deformities have developed in the femoral necks which are incompletely ossified and bent medially and caudally into an acute angle. The cartilaginous synchondroses are still widely open because of slow endochrondral bone formation at these metaphyses. The "Y" cartilages are excessive for the same reason. The crests of the tibiae are small due to undergrowth but the crests are smooth. All of the pelvic bones are too small owing to reduced growth and endochondral bone formation, and the femoral epiphyseal ossification centers are small and deformed for the same reason. The changes in pelvic bones and femora have increased with advancing age.

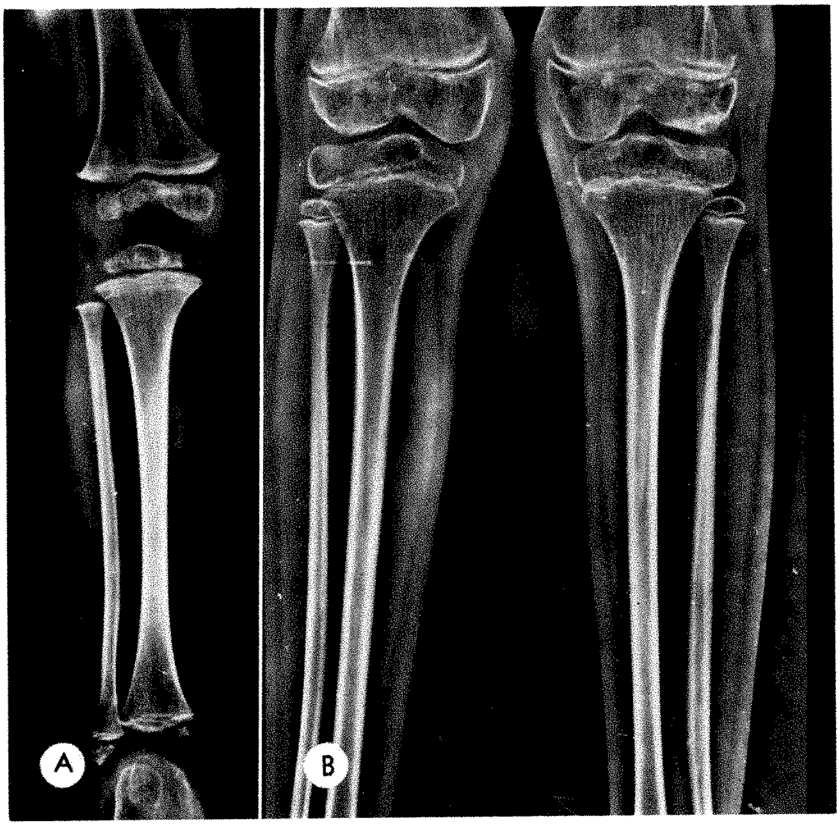

FIG. 9. Case II. (*A*) Right knee and shank at 3 years of age. All of the epiphyseal ossification centers in the femur, tibia and fibula are small, misshapen and irregularly ossified. In contrast, metaphyseal ossification of the distal end of the femur and both ends of the tibia and fibula is complete and uniform. (*B*) At 12 years, practically all of the earlier epiphyseal irregularities have disappeared and the metaphyses are normal excepting some irregularity at the distal end of the tibia. At these lower ends of the femur and at both ends of the tibia and fibula, the earlier epiphyseal lesions have disappeared with advancing age, in contrast to the changes at the proximal ends of the femur which have increased with advancing age.

and metaphyses of the tubular bones with the more marked changes in the epiphyses. The shafts of the long bones were slender with widened ends and irregular transverse lines. These changes were best seen at the hips and shoulders. In the vertebral column, the bodies in the upper thoracic levels were slightly flattened and their edges were rough. Hemivertebras at Th 5 and Th 6 produced a localized scoliosis at these levels. In the lumbar levels, the bodies were small but not flattened.

The ribs were small in caliber with spreading and cupping deformities at their sternal ends which caused the clinical costal rosary.

The calvarium was thin with retarded differentiation of the inner and outer tables. There was no evidence of dysostosis in the cartilaginous bones at the base of the skull. The pituitary fossa was normal. Dental development was normal but the upper central incisors protruded too far forward in a rodent-like position to overbite on the lower teeth.

DISCUSSION

The meta-epiphyseal and epi-metaphyseal dysostoses include a group of related but different diseases. Lamy and Maroteaux[7] termed them the micromelic form of dysplasia epiphysealis multiplex.

These patients are characterized by disproportioned growth deficiencies of such a degree that, already in early childhood, dwarfism is recognized. Children with predominance of metaphyseal changes are more severely shortened. The first signs of the disease are usually present at birth or appear during the first year of life. Later,

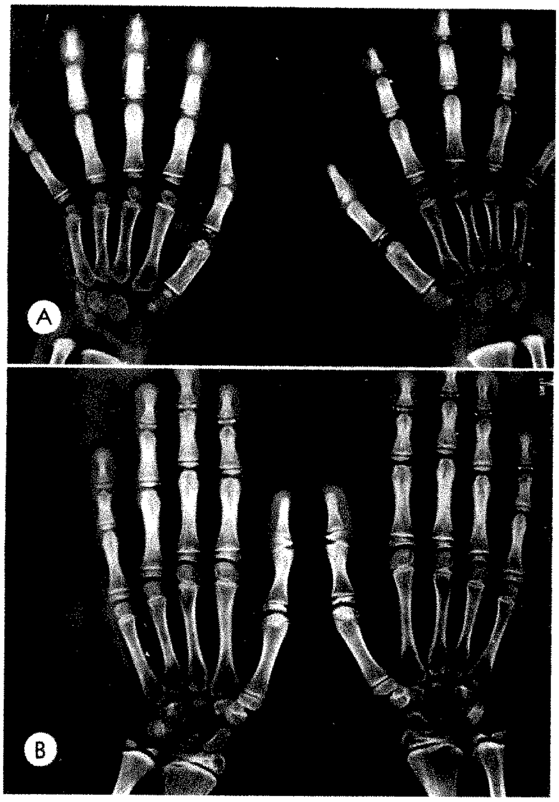

Fig. 10. Case ii. (*A*) Hands and wrists at 3 years of age. Maturation is diminished and approximates the average for a healthy girl of 2 years of age. The radial development is less than the normal 12 month level, but all of the ossification centers, excepting those in some of the proximal phalanges are smooth. (*B*) At 12 years, maturation is delayed and it approximates the average for a healthy girl 8 years of age. The ulnar epiphyseal centers are developing from 2 foci and slight angular deformities are evident in some of the round bones in the wrists. Centers in the distal phalanges of the thumb are wedge-shaped, and are hypoplastic in their lateral segments. The epiphyseal ossification centers in the distal phalanges are normal in shape but present medial and lateral sclerotic bands which represent marginal scleroses of these little bones due to incomplete formation of their medullary cavities, which have formed in the central segments of these bones but have not yet penetrated into their peripheral zones. In the hands, as at the knees, the epiphyseal changes have become more evident with advancing age. There were no metaphyseal lesions in the bones of the hands at either 3 or 12 years.

crippling deformities of the spine and extremities develop. The mental and sexual developments are normal as are all the known biochemical tests, including the level of the urine mucopolysaccharides. The roentgenographic findings may be inconclusive in early life, but later show definite changes, both at the metaphyses and epiphyses. The degree of involvement and the localization of the changes in the different metaphyses and epiphyses are different from patient to patient, to such an extent that it is difficult to find 2 closely similar examples. However, in spite of these limitations, the roentgenographic method is best, at the present time, for classification.

Case i does not fit into any group described by Catal[3] nor is it identical to any other cases of dysostosis meta-epiphysealis hitherto published. The roentgenographic changes in the pelvis of our case are very similar to those of the first case of Swoboda,[12] but other roentgenographic findings and the phenotype of the children are different.

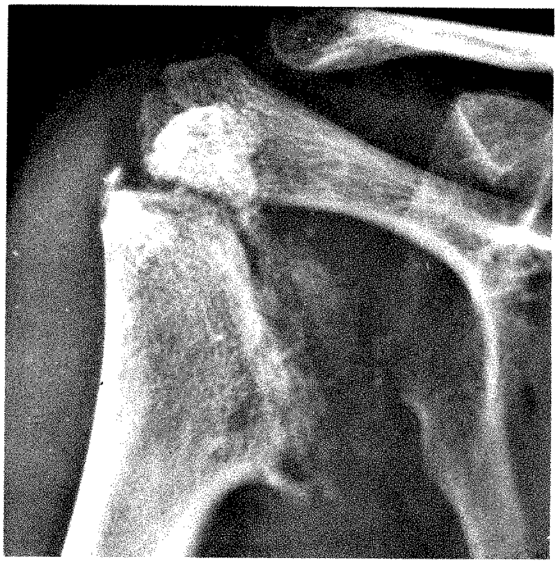

Fig. 11. Case ii. Left shoulder in frontal projection at 12 years of age. The proximal end of the humerus is enlarged and deformed. Its irregular metaphysis crosses the shaft obliquely with its medial segment more distalward, owing to undergrowth of the medial segment. The ossification center for the greater tubercle is small and regularly ossified. The ossification center for the lesser tubercle consists of a narrow irregular strip of bone which roughly parallels the oblique metaphysis.

The difficulty of classifying enchondral dysostoses is once again illustrated by our patient.

The exact diagnosis in Case II is uncertain but its classification as dysostosis epi-metaphysealis is reasonable.

The temporal changes, which are often so important in the diagnosis of skeletal dysplasias are well demonstrated in this case. A satisfactory diagnosis could not be made during early childhood because some of the crucial diagnostic changes had not yet appeared.

In this patient at the age of 3 years, multiple epiphyseal dysplasia was suggested. In the most common variety of multiple epiphyseal dysplasia—the Ribbing-Fairbank type—there are no diagnostic changes during the first 2 years of life. The difficulties in gait usually appear later, and are not as severe in the early stage as in our patient. On the basis of the roentgenographic examination, the diagnosis of multiple epiphyseal dysplasia was made at the age of 3 years, although the absence of ossification centers in the femoral heads is inconsistent with this diagnosis. At the age of 12 years, ossification of the femoral heads was still absent, but definite metaphyseal changes appeared and the diagnosis of multiple epiphyseal dysplasia could be rejected with certainty.

I would like to stress also the importance of a critical evaluation of some of the cases of dysostosis meta-epi and epi-metaphysealis. Many have not been properly identified (third case of Swoboda[12] and all the cases of Sekulowa *et al.*[11]). Others cannot be evaluated because of inadequate clinical and roentgenologic descriptions. For this reason a detailed clinical and roentgenologic account should be published with careful tracing of peculiarities of single cases. This will help to classify the similar cases into clinical groups, making possible detailed biochemical and genetic studies in the future.

SUMMARY

Two cases of combined metaphyseal and epiphyseal dysostosis are reported.

Case I, a boy, 8 years, 9 months of age had disturbance of growth and ossification in the metaphyses and epiphyses. The metaphyses and the epiphyses were affected in different degrees in different bones, but the metaphyseal changes generally predominated. The vertebral bodies were flattened above the 12th thoracic segment. The metaphyseal dysostosis was most severe at the proximal ends of the humeri, both ends of the tibiae, and distal ends of radii and ulnae. The epiphyseal dysostosis was most severe at the proximal ends of the femora and proximal ends of the humeri.

This case is difficult to classify. Currently, it should be considered an example of dysostosis meta-epiphysealis.

Case II, a female, 12 years of age, is an example of epi-metaphyseal dysostosis. Roentgenograms showed disturbances of ossification and growth in the epiphyses as well as metaphyses. The bones of the hips and shoulders were most affected. Only slight changes were present at the other epiphyses and metaphyses. This case was mistakenly called dysplasia epiphysealis multiplex at the age of 3 years. The follow-up studies at the age of 12 years showed marked change with advancing age.

The importance of caution in the diagnosis of skeletal dysplasias during the first few years of life is emphasized.

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DYSPLASIA EPIPHYSIALIS MULTIPLEX*

A CASE REPORT AND REVIEW OF THE LITERATURE

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MULTIPLE epiphysial dysplasia was first described in 1912 by Barrington-Ward.[3] Later articles describing the disease appeared in 1934 by Jansen[15] and in 1935 by Fairbank,[7] who was the first to refer to the disease as "dysplasia epiphysialis multiplex," the presently accepted term.

The majority of subsequent cases have been reported by the British. There is a low incidence of this disease in the United States. No reports have been published in the United States radiologic literature since Leeds[16] described his cases in 1960.

CLINICAL FEATURES

A hereditary familial influence is well-established.[2,11,17,19,22,23] There is no sex predilection. Jackson *et al.*[14] suggest a dominant form of inheritance with a high penetrance.

Multiple epiphysial dysplasia is asymptomatic until the age of 4 years, when gait or position disturbances occur. The presenting signs and symptoms are referable to the joint involved. Difficulty in walking, secondary to hip and/or knee involvement, is very common. There is limitation of motion and localized joint pain. The severity of the symptoms usually varies with the degree and extent of joint involvement. The roentgenographic changes in the epiphyses and joints are more marked than the clinical.

Patients are of short stature but are generally not dwarfs. Body weight may be within normal limits. The hands are stubby and the fingers blunted. The vertebral column is rarely involved.[14]

With puberty, the symptoms due to the dysplasia progressively diminish. The natural history of the disease appears to be one of self-limitation. There is usually a progressive and sometimes complete loss of original symptoms. Recovered adults, therefore, may not come to clinical attention since they often have no disability. However, early degenerative arthritis of affected joints, especially in the hips and knees, is a common sequel to the epiphysial disease. The severity of the arthritis is proportional to the epiphysial deformity.

ROENTGENOLOGIC FEATURES

There is an irregularity of multiple epiphyses which is bilateral. Surrounding the main ossification center are multiple small, irregular calcifications producing a mulberry-like appearance. Later, the epiphyses assume a more homogeneous density but may remain permanently deformed. The flattened appearance of the epiphysial center of the hips, knees, ankles, and shoulders may be permanent. The elbows, wrists, and small bones of the hands and feet are involved less commonly. The femoral head is less convex than normal and foreshortened. The acetabular and glenoid fossae are usually normal,[4] but may be irregular in conforming to the opposing articular surface of the femoral and humeral heads, respectively. Premature epiphysial fusion occurs. Coxa vara deformities are seen, probably secondary to the epiphysial involvement. Fraying and irregular mineralization of the metaphysis occur rarely.[6,9]

Wedging of the distal tibial epiphysial center is a characteristic but not a pathognomonic finding. The ankle mortise is oblique with the lateral malleolus more cephalad than the medial malleolus. This configuration may be seen in hemophilia.[6] The internal and external edges of the talus are exaggerated, producing a greater convex curve to its superior surface.

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* This material has been reviewed by the Office of The Surgeon General, Department of the Army, and there is no objection to its presentation and/or publication. This review does not imply any endorsement of the opinions advanced or any recommendation of such products as may be named.

† Chief, Department of Radiology, Kimbrough Army Hospital.

The tarsal and carpal bones may be hypoplastic. The small bones of the hands and feet are short and broad. The skull and teeth are not involved.

LABORATORY FINDINGS

No changes specific to this condition are reported. Serum calcium, phosphorus, and alkaline phosphatase are normal.

PATHOLOGY

Microscopic descriptions of the affected epiphyses are few. Anderson *et al.*[1] note features similar to those in Morquio's osteochondrodystrophy. Chondrocytes in the zone of provisional calcification are immature and reduced in number. The epiphysial line is irregular and the chondrocytes are arranged in a chaotic fashion, lacking the normal columnar arrangement. Hurley and Asling[13] have been able to produce a similar localized epiphysial dysplasia in offspring of manganese-deficient parent rats. Microscopically, spicules of osseous and marrow tissue connect the epiphysis to the metaphysis through the epiphysial plate. The epiphysial center is either absent or, when present, is misshapen, fragmented, and poorly visualized.

REPORT OF CASES

Case 1. L.P. was a 39 year old white female who first experienced episodes of pain in both knees associated with difficulty in walking at the age of 10 years. The attacks thereafter diminished in frequency and at the age of 20 years she was asymptomatic. During the previous 9 to 10 years there had been recurrences of the attacks of pain and stiffness in the knees, especially at the end of an active day. Her present difficulty began following 3 days of strenuous work. She was unable to support her weight due to severe pain in the knees.

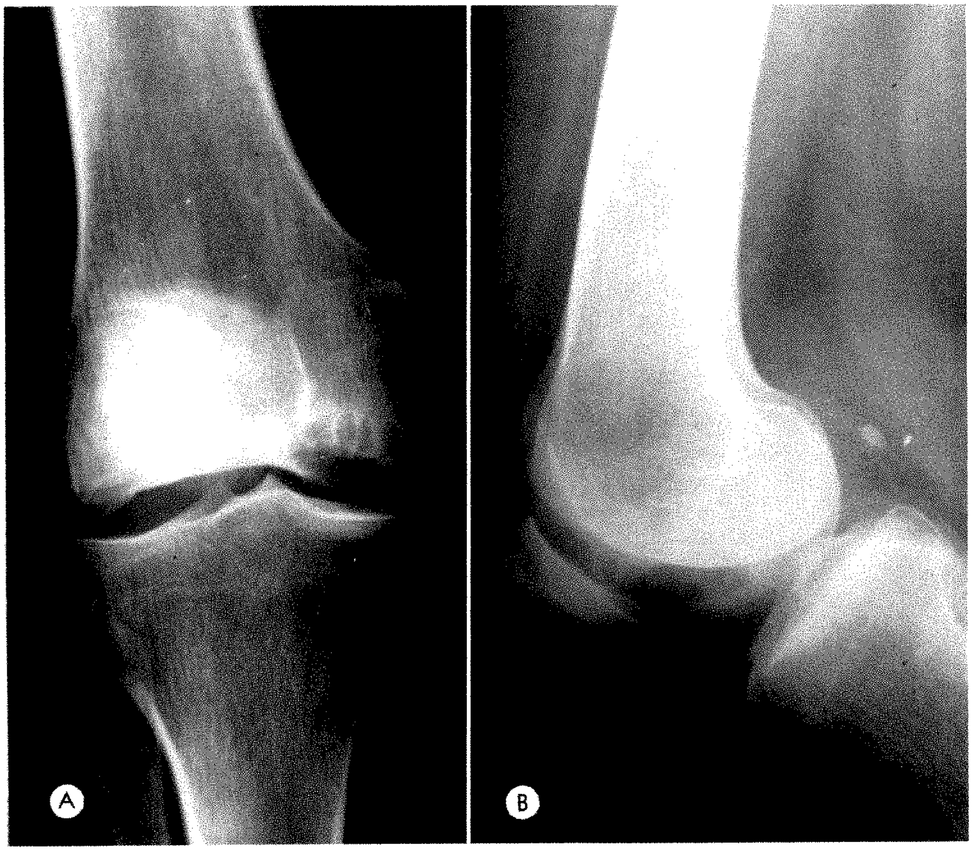


Fig. 1. Case 1. (*A* and *B*) The right knee shows flattened femoral condyles with cystic changes both medially and laterally.

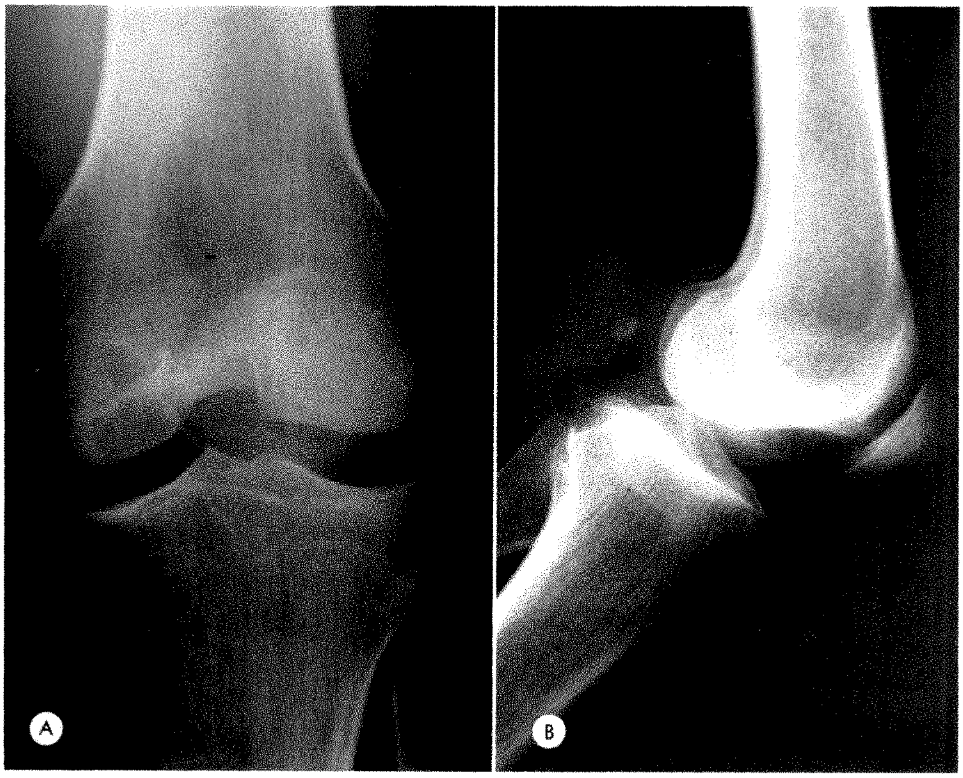
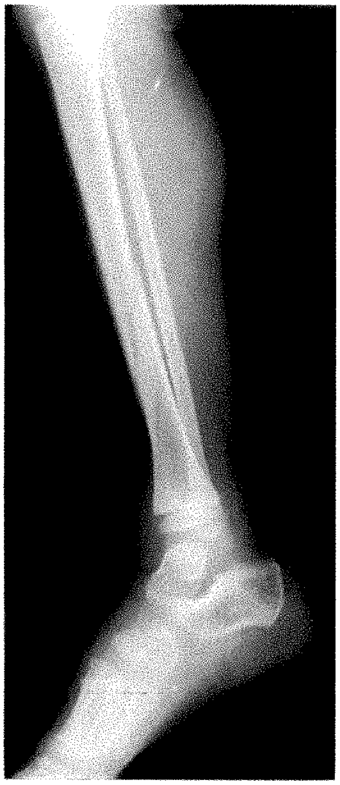


Fig. 2. Case i. (*A* and *B*) The left knee shows almost identical changes in the femoral condyles as on the right side.



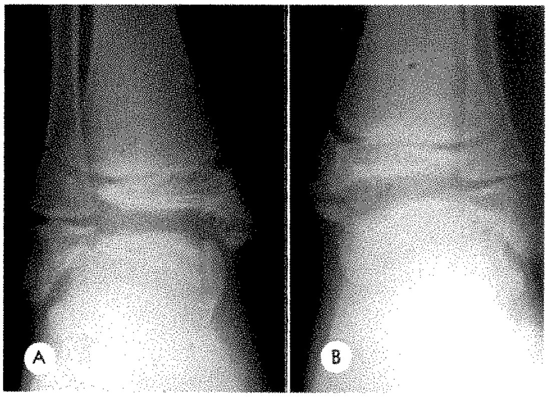


Fig. 4. Case ii. (*A* and *B*) Wedge shaped deformity of the distal tibial epiphysis producing a slanted tibiotalar joint is seen. This is more marked on the left side. Irregularity of the epiphysial center is present.

←

Fig. 3. Case ii. Epiphysial irregularity was not considered on the roentgenogram made at the time of the fracture 6 years previously.

Physical examination revealed the right knee to lock 10 degrees short of full extension. There was restricted external rotation of the leg at the knee joint. Mild atrophy of the quadriceps muscle group was present bilaterally. There was bilateral patellar-femoral crepitus and a bilateral genu valgum.

All laboratory studies including serum calcium, phosphorus, and alkaline phosphatase were normal.

Roentgenographic studies showed subchondral cysts (2 to 3 cm. in diameter) in both medial femoral condyles and smaller cysts in the lateral condyles (Fig. 1, *A* and *B*; and 2, *A* and *B*). These lesions had a moderately well-defined sclerotic border. A concave defect of the right lateral femoral condyle similar to that in osteochondritis dissecans was noted except that there was no aseptic fragment. The femoral condyle was flattened, indicative of the epiphysial disease during the active growth phase. All other joints appeared normal.

Case II. W.P. was a 12 year old white male and the oldest son of Case I. He began to walk at the age of 1 year. The mother noted the development of an abnormal gait during his fourth year, coincident with a voluntary restriction of running. A fractured left tibia (Fig. 3) was reported to have healed normally at the

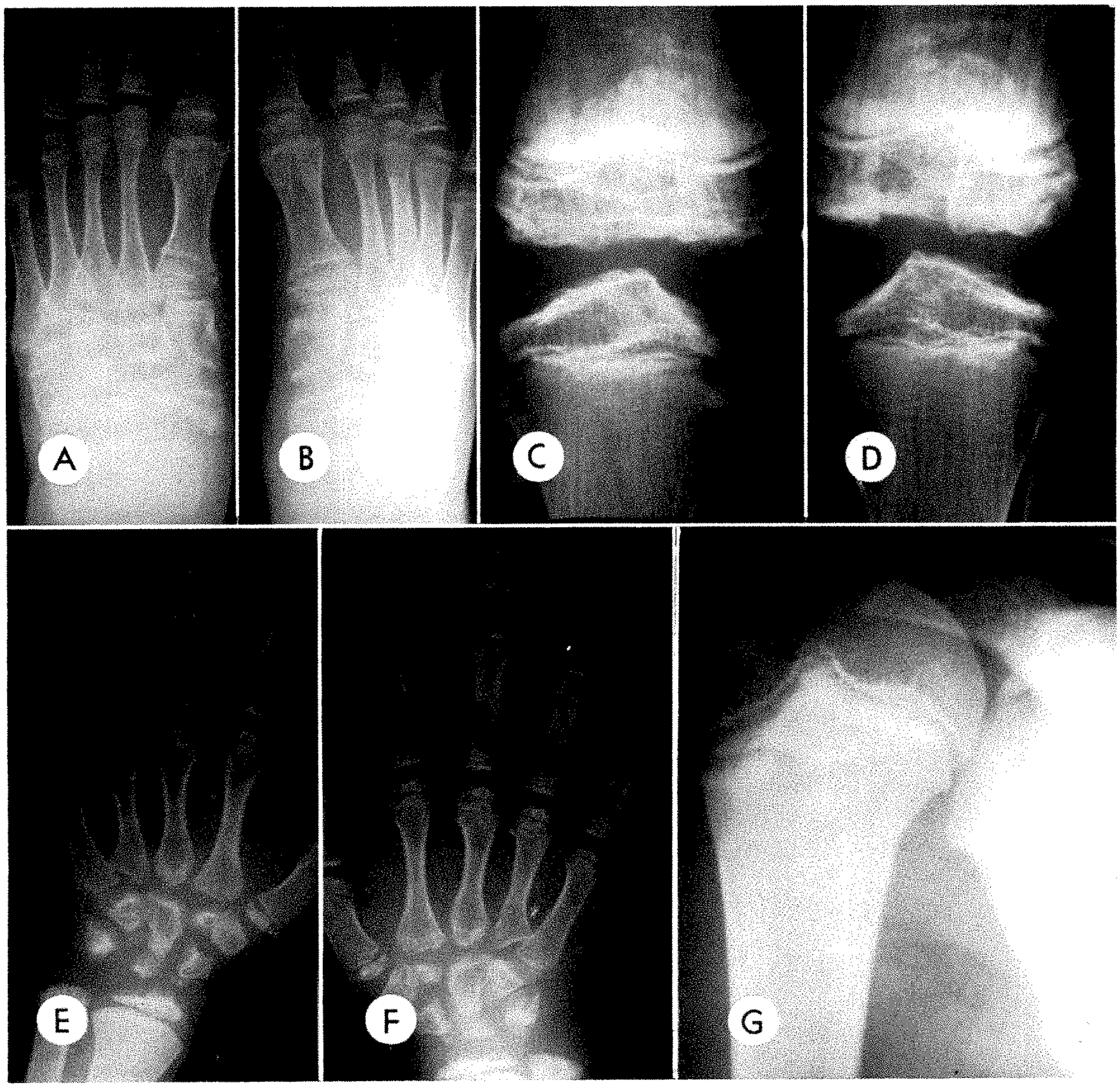


Fig. 5. Case II. (*A-G*) Polyarticular involvement is present in addition to generalized epiphysial irregularity. There is hypoplasia of the tarsals and carpals.

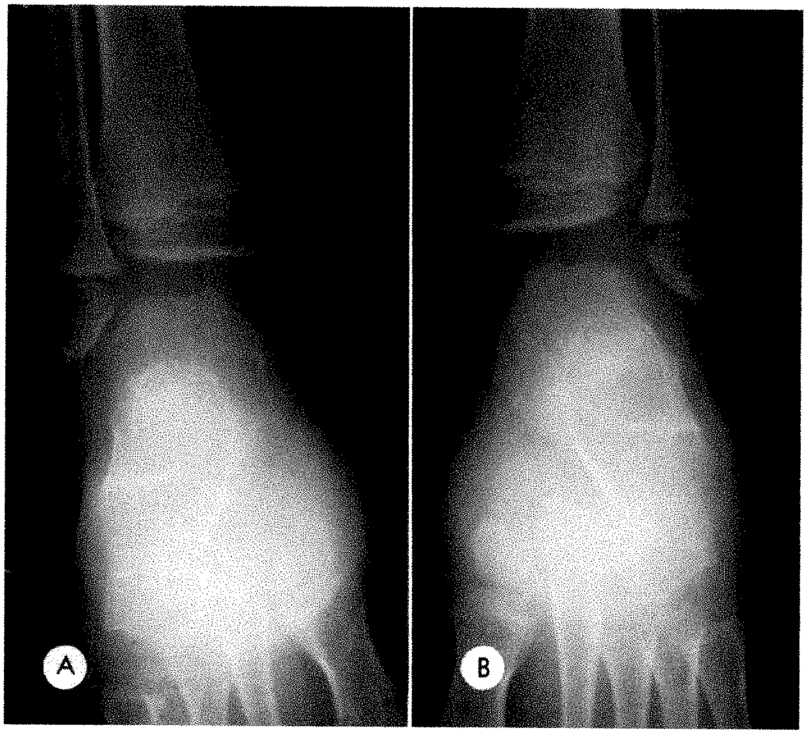


Fig. 6. Case III. (*A* and *B*) Early wedging and irregularity of the tibial epiphysis similar to Case II is present. The tarsals are also abnormal in contour.

age of 6 years. Roentgenograms taken at that time were considered otherwise normal.

Physical examination revealed hypermobile pronated feet. The other joints of the extremities were also hypermobile. The patient could not do deep knee bends but quadriceps weakness was not detected.

Serum calcium, phosphorus, and alkaline phosphatase were normal.

Roentgenograms showed wedging of the lateral aspect of the distal left tibial epiphysis resulting in a slight tibiotalar tilt (Fig. 4, *A* and *B*). There was polyarticular involvement with irregular fraying and hypoplasia (Fig. 5, *A–G*). The vertebral column was normal.

Case III. R.P. was an 8 year old white male, the youngest son of Case I and brother of Case II. He was delivered of a normal pregnancy and began walking at 13 months. A self-imposed limitation of activity during the fourth year was noted. He ran with a "clumsy" gait. There have never been swollen or painful joints.

Physical examination revealed pronated hypermobile feet. The joints were hypermobile.

Serum calcium and alkaline phosphatase were normal. The serum phosphorus was elevated to 3.2 mEq/l (normal 1.5 to 2.5 mEq/l).

Roentgenograms showed changes similar to but less marked than those in Case II (Fig. 6, *A* and *B*; 7, *A–F*; and 8). A tibiotalar tilt was developing on the left. The vertebral column was normal.

Case IV. W.S. was a 5 year old white male. At the age of 2 years, the parents noted a "pigeon-toed" gait. Legg-Perthes' disease was considered the cause.

Physical examination revealed limitation of abduction and internal rotation of the left hip. The gait was normal and there was no shortening of the legs.

Roentgenograms of the hips showed early flattening and irregularity of the proximal femoral epiphyses (Fig. 9). Irregularity of the proximal femoral metaphyses was also present. There was a slight wedging of the distal tibial epiphyses.

Serum calcium, phosphorus, and alkaline phosphatase were normal.

DISCUSSION

The roentgenographic appearance of the mother's knees (Fig. 1, *A* and *B;* and 2,

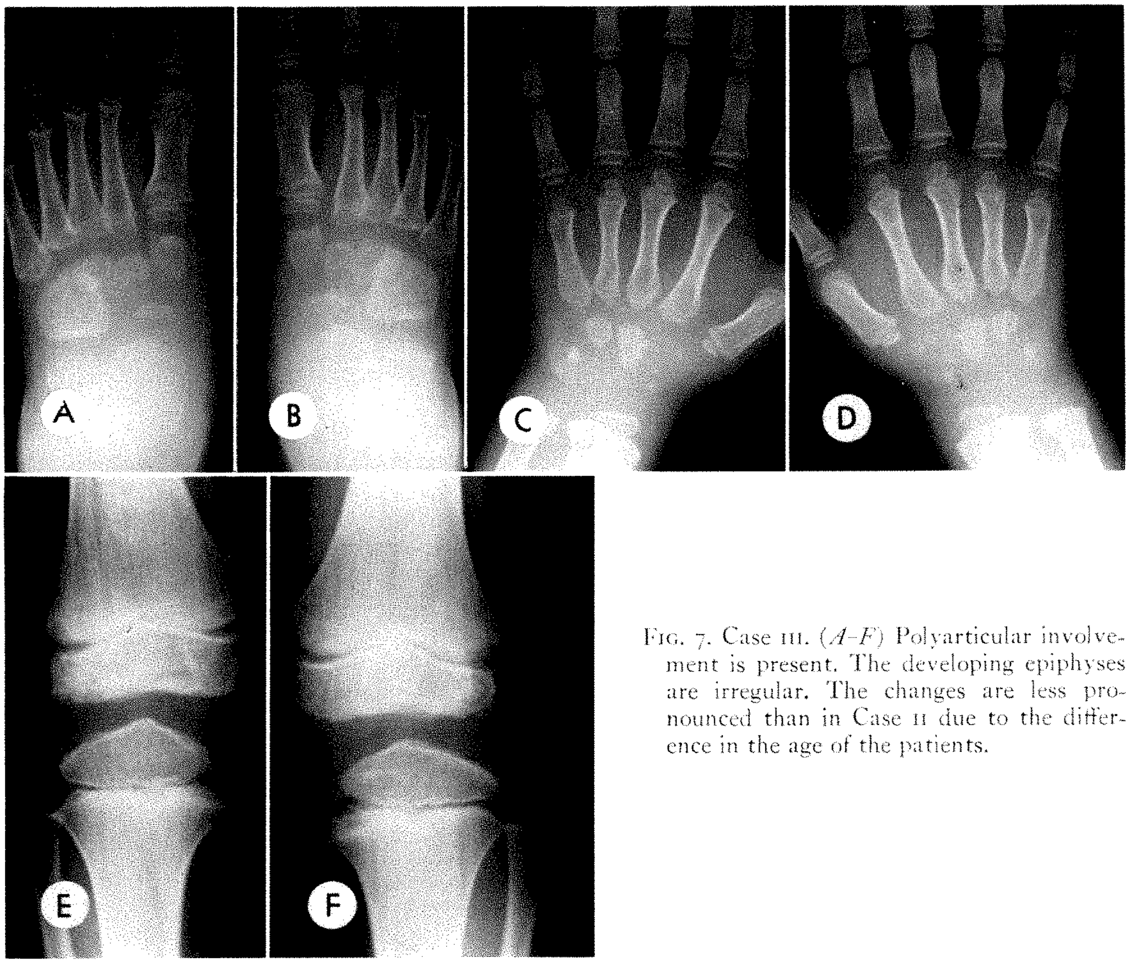


FIG. 7. Case III. (*A–F*) Polyarticular involvement is present. The developing epiphyses are irregular. The changes are less pronounced than in Case II due to the difference in the age of the patients.

A and *B*) was the first indication of a hereditary condition. This revealed flattened femoral condyles, indicating a growth disturbance in the epiphysial centers during the developmental period. There were cystic changes representing secondary degenerative arthritis and not the active Fairbank's disease itself.

Residua of epiphysial disease should be considered when flattened or abnormal condyles are present or when joint irregularities are noted, especially if bilateral.[20] Degenerative arthritis of a large joint in a young individual indicates possible previous dysplasia epiphysialis multiplex.

The wedge-shaped deformity of the distal tibial epiphyses produces a slanting of the tibiotalar joint.[8] Leeds[16] noted this deformity in 50 per cent of cases and emphasized its diagnostic importance. It was present in both Case II and Case III (Fig. 4, *A* and *B*; and 6, *A* and *B*). In children, because the distal tibial center assumes a similar appearance normally, this sign is less significant.

Not all joints of the body need be involved. Only the knees were affected in Case I. Cases II (Fig. 5, *A–G*) and III (Fig. 7, *A–F*) are characteristic of the polyarticular involvement. The hypoplastic irregular epiphyses were evident. Early irregularity of the epiphysis present at the time of the tibial fracture in Case II (Fig. 3) was similar to those just developing in Case III.

Though unproven, the roentgenographic features of Case IV (Fig. 9) suggest dysplasia epiphysialis multiplex. An original diagnosis of Legg-Perthes' disease was questioned because of the age of onset, which was much younger than is usual. The early flattening and irregularity of the femoral epiphyses were similar in appearance to the changes present in Case III (Fig. 8). This case is being followed for further evaluation and it may be that with time additional changes will become evident.[12]

DIFFERENTIAL DIAGNOSIS

1. *Legg-Perthes' disease.* This disease is confined to the hips and originates in an otherwise normal epiphysis, whereas in dysplasia epiphysialis multiplex the abnormal appearance of the involved epiphysis is seen from its inception.

2. *Cretinism.* There is femoral head flattening and a short thick femoral neck. Fragmentation of the epiphyses occurs secondary to ossification from multiple centers. The epiphyses appear late and delayed epiphysial fusion may be associated with a dense, sclerotic zone of bone in the adjacent metaphysis. The spine is affected in cretinism and rarely in dysplasia epiphysialis multiplex. Clinically, the hypothyroid features of the cretin are obvious and the laboratory findings have been well-described.

3. *Morquio-Brailsford's chondro-osteodystrophy.* The flattened, irregular, late-appearing epiphyses involve multiple joints. The first signs of the disease become manifest between the third and fourth year. The spine is always involved with a universal vertebra plana and central anterior beaking. The acetabular fossae are irregular and enlarged, uncommon in dysplasia epiphysialis multiplex.

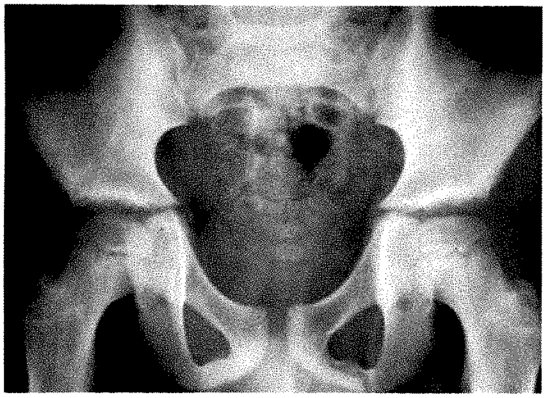


Fig. 8. Case III. There is flattening of the left femoral epiphysis accompanied by irregularity of the metaphysis. The acetabular roof is less concave than on the right side. There is irregularity of the meatphysis.

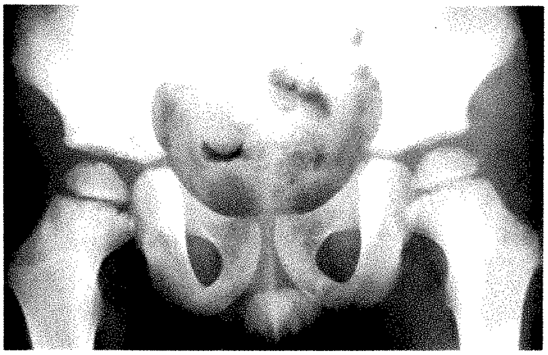


Fig. 9. Case IV. The flattening and irregularity of the femoral epiphyses are similar to Legg-Perthes' disease. Dysplasia epiphysialis multiplex seems more likely than Legg-Perthes' disease because of the age of onset.

4. *Hurler's disease.* The clinical appearance of the gargoyle is recognized by the age of 2 or 3 years. Mental retardation differentiates this disease from dysplasia epiphysialis multiplex in which mental development is normal. The epiphysial centers are small and irregular but are less fragmented. The acetabular fossae are irregular and widening of the diaphysis of the tubular bones is present. Tapering of the ribs, shallow glenoid fossae, swollen clavicles, and vertebral deformities are absent in dysplasia epiphysialis multiplex.

5. *Achondroplasia.* This congenital disorder is recognized at birth. The epiphysial ossification centers are small and appear late. Though slightly abnormal in shape, they are not irregular. Flaring of the distal shafts of the tubular bones and ribs is a distinguishable point. The hands and feet are broad and stubby and there is shortening of the tubular bones. The tapering of the lumbar spine inferiorly and narrowing of the lumbar canal are characteristic.

6. *Epiphysial dysplasia punctata.* This disease is congenital and fatal before the end of the first year of life. The entire epi-

physial center ossifies from multiple discrete centers unlike the satellite epiphysial calcifications of dysplasia epiphysialis multiplex. Bilateral congenital cataracts are seen in 50 per cent of cases.

SUMMARY

1. The clinical and roentgenologic features of dysplasia epiphysialis multiplex are presented.

2. Bilaterally flattened condyles in the adult may be the residua of previously unrecognized Fairbank's disease.

3. The unusual early onset of degenerative arthritis may be secondary to epiphysial damage produced by dysplasia epiphysialis multiplex during childhood. Children of affected parents should be examined roentgenographically for early recognition of the disease in order to initiate treatment promptly.

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AN UNDIAGNOSED BONE DYSPLASIA*

A 2 FAMILY STUDY OF 4 GENERATIONS AND 3 GENERATIONS

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A 3 YEAR old white female was admitted to the hospital for evaluation because of a failure to thrive. Roentgenograms of the chest and abdomen showed a severe bone abnormality. This finding led to a complete bone survey of the entire family, which included 4 generations (Chart I). The youngest was 16 months of age and the oldest was 80 years of age. Some degree of involvement was found in 9 of the 14 members of the family.

A second family was studied consisting of 6 members and representing 3 generations (Chart II). Three members of this family, representing 2 generations, showed severe involvement with the syndrome. Photographs of a fourth member suggested bone involvement on the basis of the facial characteristics. This patient represented the third generation.

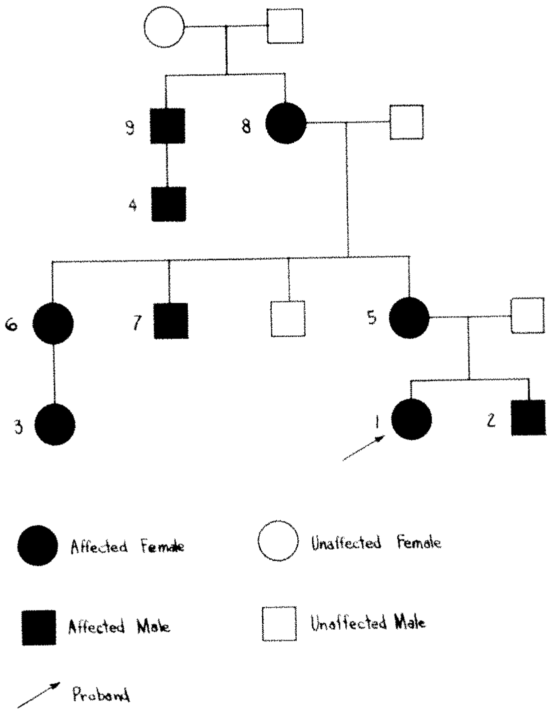

Chart I. Family I.

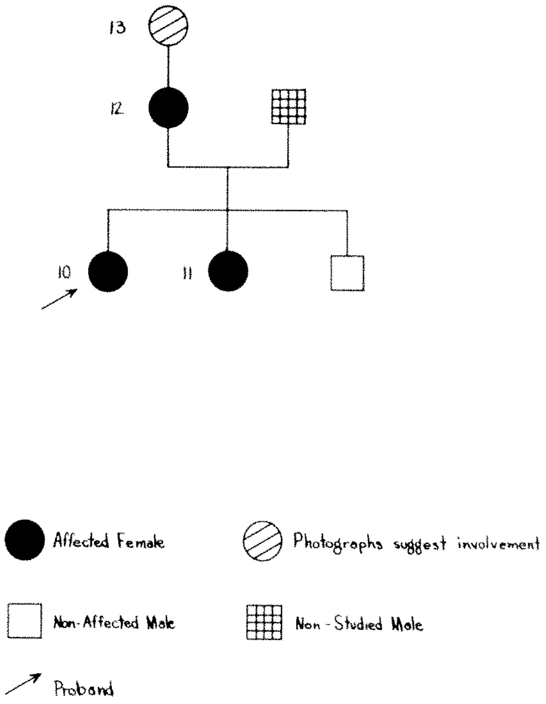

Chart II. Family II.

Table I gives the genealogy of the 2 families.

The disorder in both families appeared to fulfill the definition of a bone dysplasia in that the predominant error was intrinsic to the bone, it affected all bones similarly and was generalized in distribution. A review of the available literature failed to reveal a description of a similar disease. Thus, it was felt that the dysplasia was sufficiently rare to warrant a report.

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TABLE I

Case No.	Age (yr.)	Sex	Relationship	Generation
		FAMILY I		
1	3	F	proband	F iv
2	16 mo.	M	brother	F iv
3	22 mo.	F	first cousin	F iv
4	11	M	third cousin	F iii
5	24	F	mother	F iii
6	22	F	aunt	F iii
7	19	M	uncle	F iii
8	48	F	grandmother	F ii
9	47	M	granduncle	F ii
		FAMILY II		
10	3	F	proband	F iii
11	3 mo.	F	sister	F iii
12	34	F	mother	F ii
13	59	F	grandmother	F i

FAMILY I

CASE I

Case 1, a female, aged 3 years, F iv, propositus, was the original case studied in the first family. She was the product of a full-term gestation, with a birth weight of 5 pounds, 14½ ounces. Numerous observations of weight, length and circumferences of the head and chest over the 3 year period showed that these were considerably below the 50th percentile according to Wetzel's normograph. The child had numerous respiratory and ear infections. She was frail, delicate and underweight. The gait was abnormal. The teeth showed malalignment. The ears were large and the eyes prominent.

Laboratory Findings. The sweat test, serum protein, albumin, globulin, albumin/globulin ratio, calcium, alkaline phosphatase, acid phosphatase, complete blood cell count and urinalysis were normal. The inorganic phosphorus was slightly elevated on 2 occasions, 4.5, and 4.3 mg. per cent, respectively (the normal range is 2.7–4.0

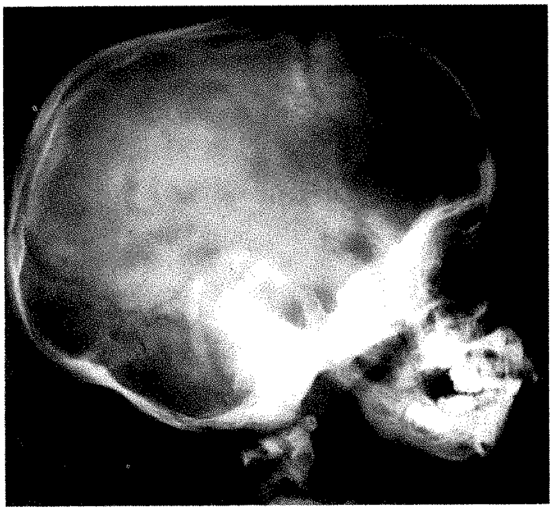

FIG. 1. Case 1. Lateral roentgenogram of skull. The anterior fontanelle is open. A micrognathia with a marked overbite is present. The base of the skull and mastoid processes are sclerotic.

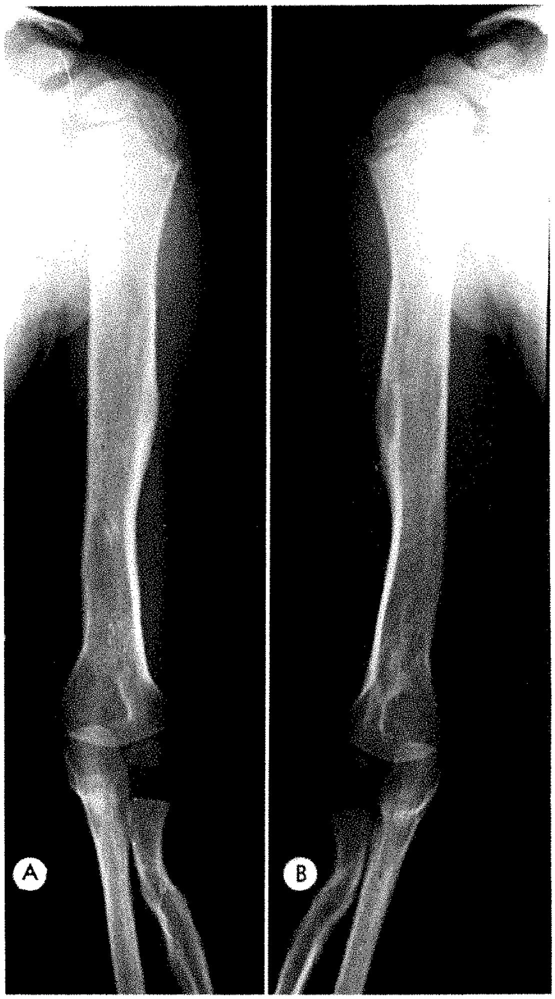

FIG. 2. Case 1. (*A* and *B*) Anteroposterior roentgenograms of humeri. Bowing and cortical irregularity are evident in the radii. The humeri show under cylindrization.

mg. per cent). The karyotype was normal.

Roentgenographic Findings.

Skull. The anterior fontanelle was open. The base of the skull and the mastoid processes showed a moderate sclerosis. The paranasal sinuses were underdeveloped. Micrognathia was present and produced a marked overbite (Fig. 1).

Long Tubular Bones. There was flaring of the metaphyses at the proximal and distal ends of the humeri, femora and tibiae. The radii, ulnae and fibulae were only slightly flared. Marked bowing of the radii and tibiae produced an "S" shaped appearance in the anteroposterior roentgenogram. Cortical irregularity was generalized. A severe coxa valga was present with the femoral necks vertical to the shafts of the femora (Fig. 2, *A* and *B*; 3; and 4).

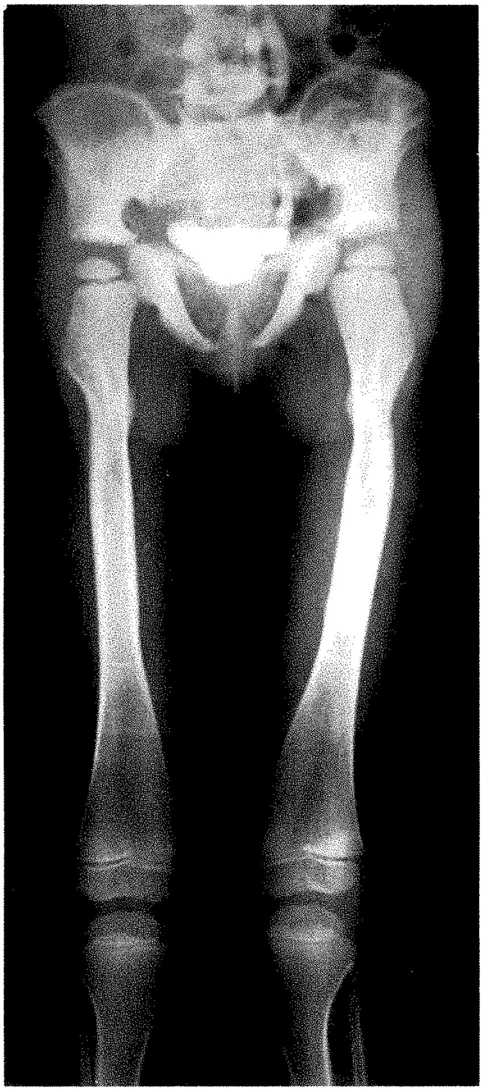

Fig. 3. Case 1. Anteroposterior roentgenogram of pelvis and femora. A hydroureter is present on the left. The iliac bones are flared at the crests and flattened in the area of the acetabula. Coxa valga and metaphyseal flaring are present in the femora. The ischial bones are tapered.

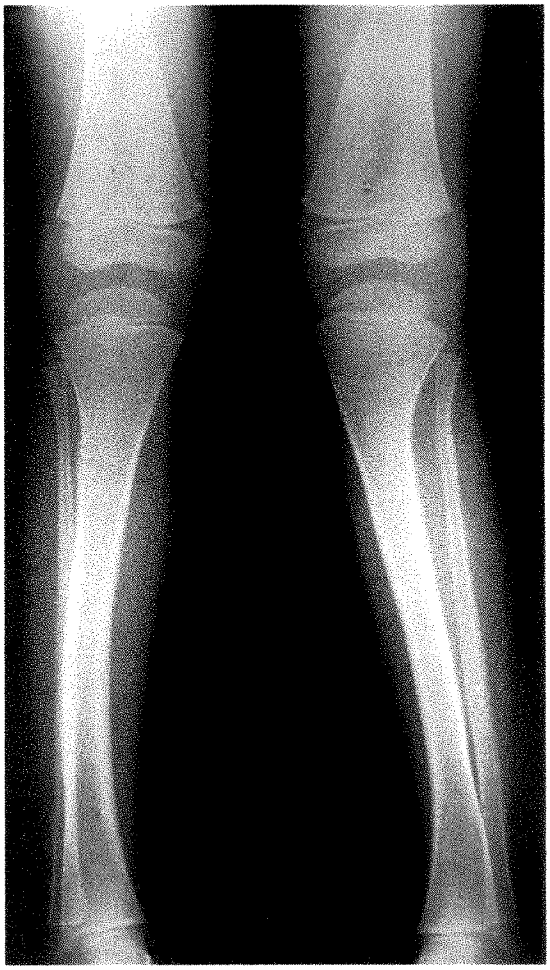

Fig. 4. Case 1. Anteroposterior roentgenogram of knees and tibiae. The tibiae present an "S" shaped appearance. The metaphyses are flared. Both fibulae are bowed.

Short Tubular Bones. There was shortening of many phalanges of the hands (Fig. 5, *A* and *B*) and feet. This was most marked in the distal phalanges of the thumbs and great toes.

Wrists, Sternum and Vertebrae. The dis-

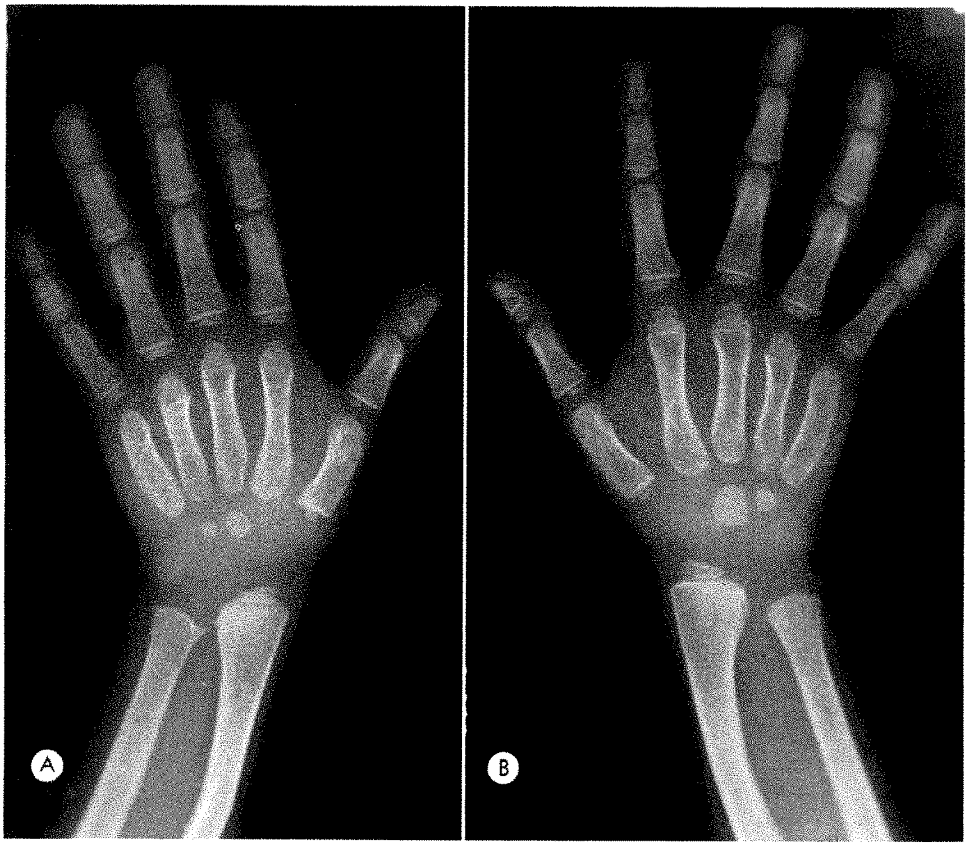

Fig. 5. Case 1. (*A* and *B*) Anteroposterior roentgenograms of hands and wrists. The distal phalanges of the thumbs are shortened. The radii are bowed. Lack of normal cylindrization is present in several metacarpals.

tal ends of the radii and ulnae were tilted towards each other (Fig. 5, *A* and *B*). Only 2 ossification centers were present in the sternum; 1 for the manubrium and 1 for the superior segment of the gladiolus. The vertebral bodies in the lower thoracic and the upper lumbar areas were increased in height.

Flat Bones. The scapulae were shortened in their vertical diameter. The iliac bones were flattened in the area of the acetabulum and flared at the crest. The ischia were tapered. The bodies and inferior rami of the os pubis were not mineralized. The superior rami were small and attenuated (Fig. 3).

Ribs. The ribs revealed multiple deformities. Irregular constrictions and contours produced a ribbon appearance (Fig. 6).

Urinary System. There was a hydronephrosis and hydroureter on the left side with distal ureteral stenosis (Fig. 3).

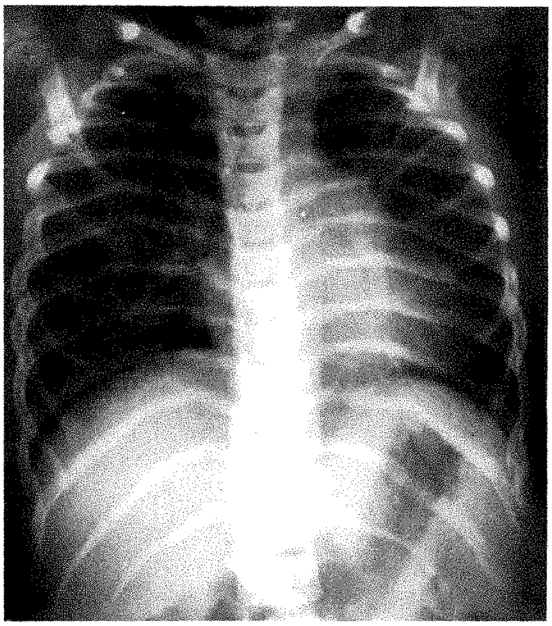

Fig. 6. Case 1. Anteroposterior roentgenogram of ribs. Marked deformity with cortical irregularity is present. The ribs give a ribbon appearance.

Other Bones. The clavicles were narrowed and irregular laterally (Fig. 6).

Teeth. There was malalignment of all the teeth.

Comment. A 16 month old male (Case 2, F iv), brother of Case 1 and a 22 month old female (Case 3, F iv), cousin of Case 1, demonstrated similar clinical and roentgenographic findings.

An 11 year old male (Case 4, F iii), cousin of Case 1, had a moderate overbite and malalignment of the teeth but no other stigmata of the disease. The patient has been treated for coxa vara with braces for several years.

CASE 5

The mother (Case 5, F iii) of Case 1 presented the adult form of the dysplasia.

Laboratory Findings. The blood chemistries and karyotype were normal.

Roentgenographic Findings.

Skull. A moderate degree of hyperostosis interna frontalis was present on the right side. There was sclerosis of the base of the skull. The greater wing of the left sphenoid was widened. The frontal sinuses were poorly developed.

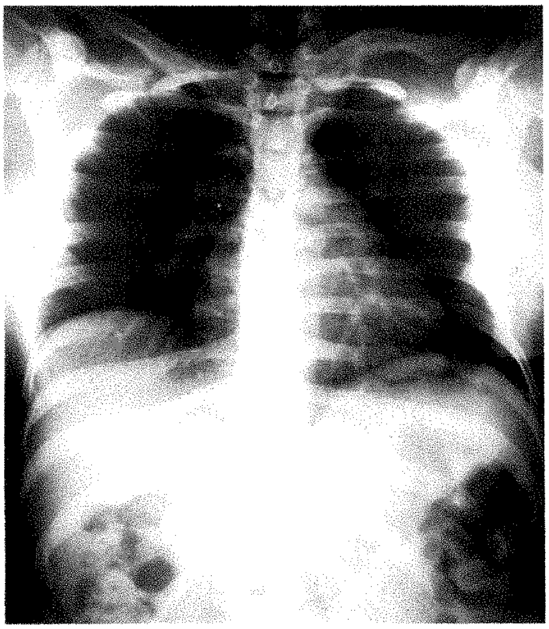

Fig. 7. Case 5. Anteroposterior roentgenogram of ribs. The ribs are narrowed posteriorly and broadened laterally and anteriorly. The twelfth ribs show cortical irregularity.

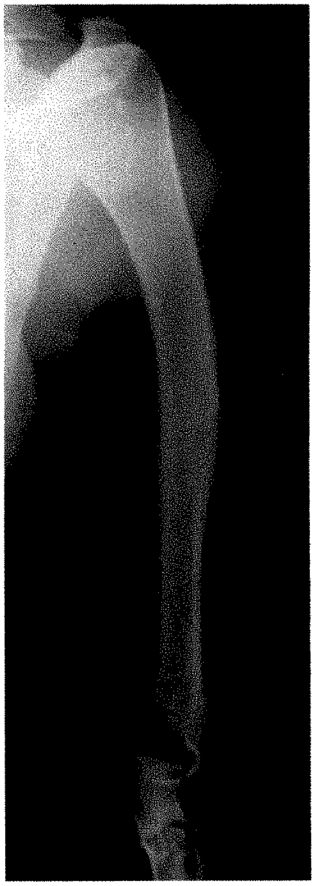

Fig. 8. Case 5. Anteroposterior roentgenogram of left humerus. The proximal shaft is bowed laterally.

Mandible. Micrognathia produced a moderate overbite.

Ribs and Clavicles. There was widening of the clavicles medially. Many of the ribs were narrowed posteriorly with prominent flaring of their anterior portions. This was particularly marked in the lower ribs. Irregularity of the twelfth ribs produced a ribbon appearance (Fig. 7).

Long Tubular Bones. A mild degree of abnormal cylindrization of the proximal tibiae and distal right femur was present. The left humerus was slightly longer than the right humerus and had a marked lateral bowing of the proximal portion of the shaft (Fig. 8).

Vertebrae. The lumbar vertebral bodies were increased in height with narrowed

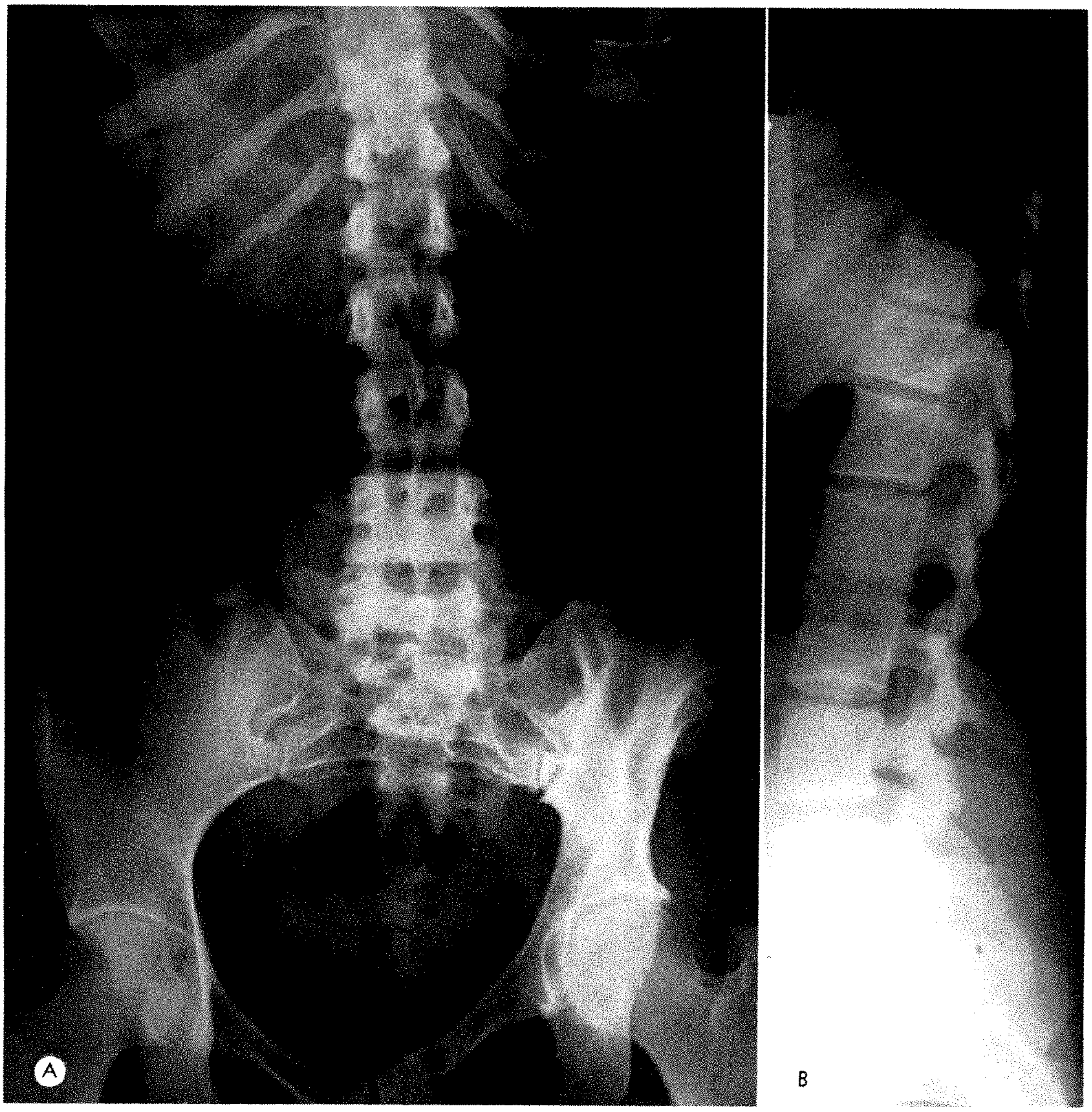

Fig. 9. Case 5. (*A*) Anteroposterior roentgenogram of lumbar spine and pelvis. The right innominate bone is hypoplastic. The lower ribs show cortical irregularity and deformity in contours. (*B*) Lateral roentgenogram of lumbar spine. The lumbar vertebrae are increased in height.

intervertebral disk spaces between the vertebrae (Fig. 9, *A* and *B*).

Flat Bones. The right innominate bone was hypoplastic and irregularly shaped (Fig. 9*A*).

Comment. An aunt (Case 6, F iii) of Case 1 had a minimal overbite. Several upper ribs were narrowed posteriorly. The proximal phalanges of the second, third and fourth toes were shortened in both feet. The pelvis and tubular bones were not remarkable.

An uncle (Case 7, F iii) of Case 1 had a hypoplastic mandible resulting in a moderate overbite. There were under cylindrization and cortical irregularity of the right femur and humerus. A moderate hypoplasia involved the right pubic bone and ischium. The right obturator foramen was small. The ribs were markedly widened throughout their lengths with the exception of narrowing near their vertebral attachment. There was widening of the medial ends of the clavicles.

The grandmother (Case 8, F ii) of Case 1 had a moderate overbite. The proximal phalanges of the second and third toes were shortened in both feet. The lumbar vertebrae were increased in height.

A granduncle of Case 1 (Case 9, F ii) had a slight overbite. The first metatarsals were shortened. The remaining bones were normal.

FAMILY II

CASE 10

The roentgenographic changes in a 3 year old female (Case 10, F iii), propositus of Family II, could almost be superimposed on those of Case 1, Family I (Fig. 10, 11 and 12).

Comment. A sister (Case 11, F iii) of Case 10 was first observed *in utero* at 6 months of gestation. Roentgenographic findings were present in the ribs and long tubular bones (Fig. 13).

Roentgenograms at 3 months of age showed a micrognathia with a mild overbite. Generalized cortical irregularity was present in the tubular bones. There was flaring of the metaphyses of the humeri, radii, fibulae and tibiae. Bowing of the tibiae produced an "S" shaped appearance. The proximal tibial epiphyses were tilted

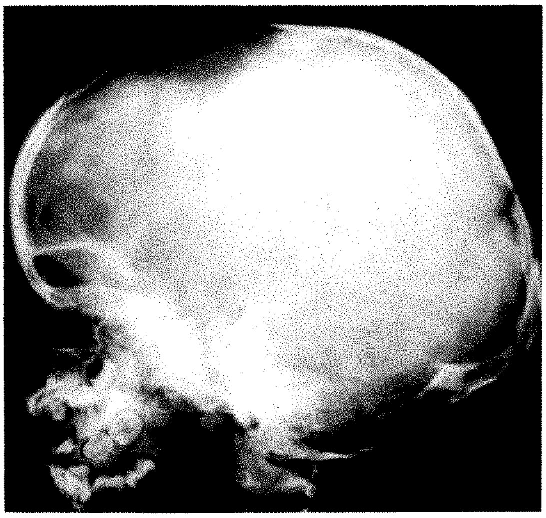

Fig. 10. Case 10. Lateral roentgenogram of skull. The anterior fontanelle is open. A micrognathia with an overbite is present. Sclerosis is present in the skull and mastoid processes. Compare with Figure 1.

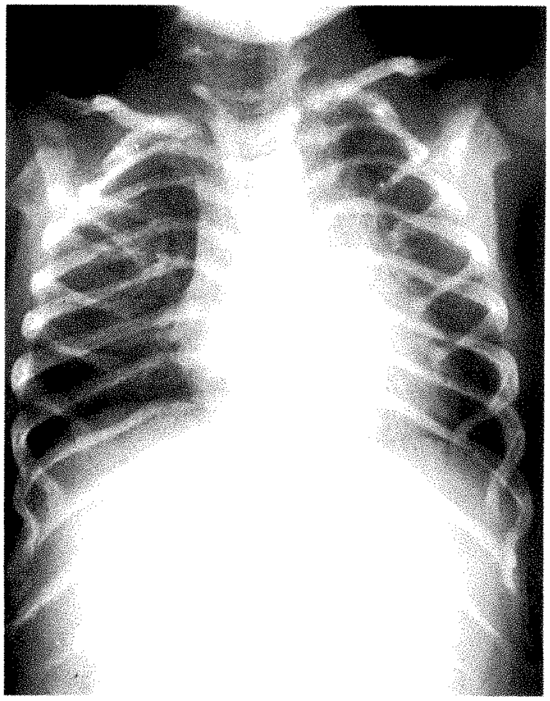

Fig. 11. Case 10. Anteroposterior roentgenogram of ribs. The ribs are deformed. Cortical irregularity is generalized. A ribbon appearance is present. Compare with Figure 6.

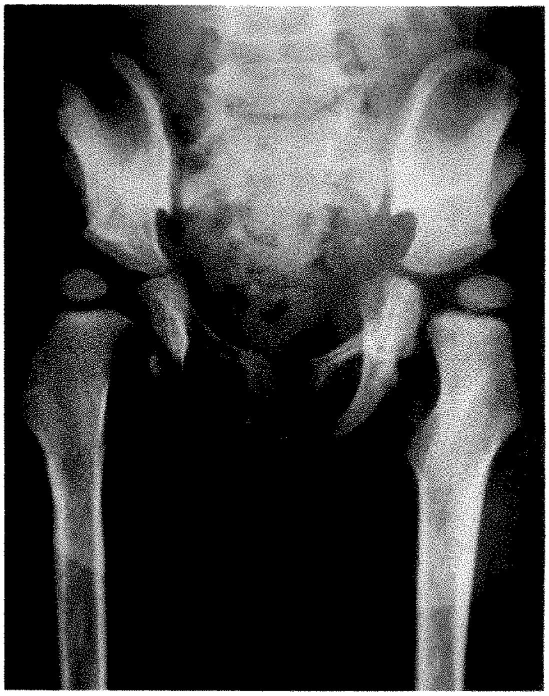

Fig. 12. Case 10. Anteroposterior roentgenogram of pelvis. The iliac crests are flared. The femora show a coxa valga deformity. Compare with Figure 3.

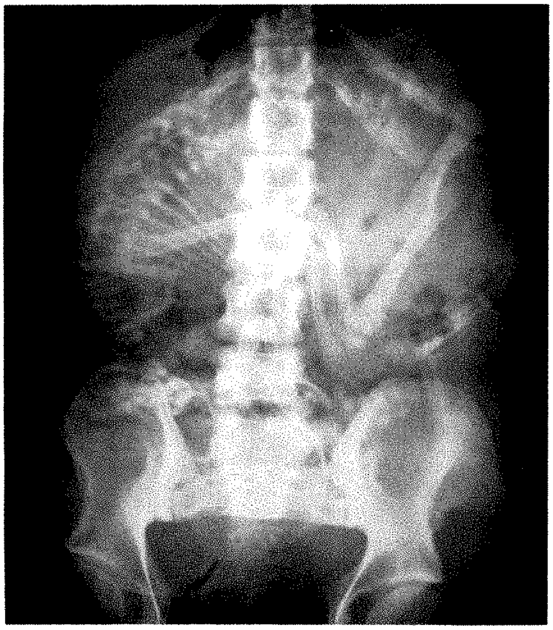

FIG. 13. Case 12. Anteroposterior roentgenogram of abdomen and pelvis. Case 11 *in utero* at 6 months' gestation. Rib and long bone changes are present. The maternal pelvis shows iliac crest flaring and increase in the height of the lumbar vertebrae. The ribs have an irregular contour.

laterally. The distal phalanges of the toes were shortened. A concavity in the central portion of the lower thoracic and lumbar vertebral bodies produced a double beak appearance. There was a delay in the ossification of the sternum. The ribs had multiple areas of irregular narrowing. There was flaring of the iliac bone on the right.

CASE 12

The mother (Case 12, F ii) of Cases 10 and 11 had a moderate degree of hyperostosis interna frontalis bilaterally. Sclerosis was present in the base of the skull and mastoid processes. The metaphyses of the humeri, femora and tibiae showed flaring. There were under cylindrization and bowing of the tibiae and fibulae. Coxa valga was present. The lower thoracic and upper lumbar vertebral bodies had an anterior concavity, producing a double beak appearance similar to that seen in Case 11. Six lumbar vertebrae were present. There was flaring of the iliac wings (Fig. 13). The superior rami of the os pubis were narrowed. The scapulae were small and showed constriction of the bodies. The ribs were narrowed near their attachment with the vertebral column and broadened laterally and anteriorly. The cortices were thinned throughout the length of the ribs. There was flaring of the ends of the clavicles with irregular areas of constriction centrally.

Comment. Photographs of the grandmother (Case 13, F i) of Case 10 strongly suggested the presence of the bone dysplasia in the skull, mandible and long tubular bones.

DISCUSSION

Table III gives the blood chemistry values in Cases 1, 10, 11 and 12 and the urine analysis values in Cases 10 and 11.

TABLE II

ROENTGENOGRAPHIC MANIFESTATIONS

Skull
- a) delay in closure of anterior fontanelle
- b) sclerosis of base and mastoids

Mandible
- a) micrognathia
- b) overbite
- c) malalignment of teeth

Tubular Bones
- a) metaphyseal flaring
- b) bowing—"S" shaped appearance of tibia
- c) shortening
- d) cortical irregularity
- e) coxa valga

Ribs
- a) cortical irregularity
- b) ribbon appearance

Vertebrae
- a) increase in height
- b) anterior concavity with double beak
- c) decreased disk spaces in lumbar area

Flat Bones
- a) flaring of iliac crests
- b) narrowed obturator foramen
- c) tapered ischia

Other Bones
- a) delay in sternal ossification
- b) cortical irregularity of clavicles
- c) flaring of clavicles

Numerous laboratory studies in Cases 10, 11 and 12, Family II, were performed. The results were normal with the exception of a slightly elevated serum phosphorus in Case 11. A similar finding was observed in Case 1, Family I.

Table III

BLOOD CHEMISTRIES

	Case 1	Case 10	Case 11	Case 12
Calcium (mg. %)	10.1 9.3	10.2	10.8	9.6
Phosphorus (mg. %)	4.5 4.3	5.0	5.8	3.1
Alkaline phosphatase	13.9* 15.2*		7.2†	1.1†
Blood urea nitrogen (mg. %)	14	13	16	14
Glucose (mg. %)	60	98	104	96
Protein (gm. %)	7.98	7.0	6.1	6.4
Albumin (gm. %)	4.23	4.4	4.0	3.6
Cholesterol (mg. %)		124	236	180
A/G ratio	1.13			
Globulin	3.75			

* King-Armstrong units.
† Bodansky units.

URINE ANALYSIS

	Case 10	Case 11
Creatinine (mg./L)	533	355
Calcium concentration (mMol./L)	5.15	1.50
Phosphorus concentration (mg./L)	1370	800
Free hydroxyproline concentration (mg. %)	less than 1	less than 1
Total hydroxyproline concentration (mg. %)	14.5	9.4
Total alpha amino nitrogen (μMol./mg. creatinine)	normal	normal
Amino acid chromatogram	normal	normal

Table IV

PERTINENT FEATURES

1. familial (hereditary?)
2. congenital
3. female predominance (?)
4. characteristic facies
   a) exophthalmos
   b) full cheeks
5. micrognathia
6. malalignment of teeth
7. normal karyotype
8. slightly elevated serum phosphorus*

* Case 1 and Case 11.

The roentgenographic manifestations encountered in the bones of the affected members of the two families are listed in Table II.

The ethnic origin of the 2 families was sufficiently different to exclude a close relationship. The father of Case 1, Family I, was Hungarian. The remaining members of the family were of Italian origin. All members of Family II were of Anglo-Saxon origin.

In certain areas there were similarities to other individual bone dysplasias; however, the over-all pattern was so different that it was felt that this was a new entity.

The pertinent features characteristic of the entity described are enumerated in Table IV.

SUMMARY

A severe bone abnormality was observed in a 3 year old white female which led to a family study of 4 generations. Nine of the 14 members of the family revealed some degree of involvement. The most severe changes were found in the original case (Case 1) and the mother (Case 5). The youngest patient was 16 months of age.

A second family, representing 3 generations, presented 3 similar cases (Cases 10, 11 and 12) and possibly a fourth case (Case 13). The youngest patient was observed *in utero*.

A total of 18 members of the 2 families

was studied. Thirteen of these, 9 females and 4 males, showed some degree of bone involvement. The roentgenographic and clinical findings were practically identical in both families.

The disorder is presented as a possible new entity among bone dysplasias.

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THE ROENTGEN APPEARANCE OF PSEUDOHYPOPARATHYROIDISM (PH) AND PSEUDO-PSEUDOHYPOPARATHYROIDISM (PPH)*

DIFFERENTIATION FROM OTHER SYNDROMES ASSOCIATED WITH SHORT METACARPALS, METATARSALS, AND PHALANGES

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MUCH confusion is apparent in reports of the significance of short or disproportionately short metacarpals, metatarsals, and phalanges in the diagnosis of pseudohypoparathyroidism (PH) and pseudopseudohypoparathyroidism (PPH). Van der Werff ten Bosch[65] concluded that the only abnormalities patients with PPH had in common were reduced stature and brachydactylia. Brachydactylia was attributed to a developmental abnormality of the metacarpal and metatarsal bones. In his report, van der Werff ten Bosch[65] included 4 cases of gonadal dysgenesis with 4 of classic PPH in a syndrome he called "brachymetacarpal dwarfism." Roche,[52] Todd, Hill, Nickerson, and Tingley,[62] and Uhr and Bezahler[64] reported instances of multiple familial exostosis with short metacarpals as PPH. Bell, Gerard, and Bartter,[10] Costello and Dent,[21] and Singleton and Teng[55] have described patients with PH whose stature, hands, and feet were normal, but who had superimposed secondary hyperparathyroidism.

METHOD

In this report the roentgen findings of 57 patients with PH and 20 with PPH reported in the literature are analyzed. Our own 3 patients, previously reported,[33,58] are included. Only those were selected whose roentgenograms of the hands were described in detail, or were adequately reproduced for interpretation. From reviewing these cases, we have concluded that the roentgen descriptions are frequently incomplete, with attention focused upon disproportionate shortening of the metacarpal and metatarsal bones. Those roentgenograms showing uniform shortening of the metacarpals were usually reported as normal. Also, many obvious anomalies of the phalanges were not discussed in the text. For this reason, our tabulated results (Tables I to X) represented only the minimal roentgen manifestations; the actual abnormalities are probably considerably greater. Our own evaluation of 3 patients is based on a roentgen survey of all important skeletal regions. The following definitions were used for selection.

PH is a congenital, hereditary abnormality characterized by hypocalcemia and hyperphosphatemia, as in hypoparathyroidism. It does not, however, respond adequately to parathyroid hormone. The parathyroid glands are present and may be hyperplastic, but the hormones produced by them lack the ability to effect adequate phosphate diuresis. Most of these patients are short, obese, and mentally retarded, and corneal or lenticular opacities and brachydactylia may be present.

Roentgenograms of many show calcification or ossification of the skin or subcutaneous tissues, or calcification of the basal ganglia or other parts of the brain. The roentgen studies may also give evidence of exostosis, accelerated closure of epiphyses, short phalanges, metacarpals and metatarsals, and delayed or defective dentition.

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† The views expressed herein are those of the author and do not necessarily reflect the views of the Air University, the United States Air Force or the Department of Defense.

TABLE I

SKULL ABNORMALITIES NOTED IN PUBLISHED CASE REPORTS OF BOTH PH AND PPH AND IN OUR PATIENTS WITH PH*

	PH				PPH	
	Published Case Reports (no.)	Our Patients (no.)	Total	Per Cent	Published Case Reports (no.)	Per Cent
Cases reviewed	57	3	60		20	
Skulls examined roentgenologically	49	3	52		12	
Abnormalities						
Thickened calvarium or widened diploe	18	1	19	37	3	25
Thick inner table	1	0	1	2	3	25
Basal ganglia calcification (including dentate nucleus)	22	1	23	44	1	8
Other intracranial calcification	8	0	8	15	1	8
Small pituitary fossa	3	0	3	6	0	0
Brachycephaly	2	1	3	6	0	0
Abnormal dentition	19	0	19	37	0	0

* Data obtained from references: 1–4, 6–8, 10, 12, 13, 15, 16, 18, 20–31, 33–39, 41–43, 45, 46, 48–51, 55, 57, 59, 61, 63, 66.

In some instances, the skeletal density is diffusely decreased, usually ascribable to osteoporosis. In others, the typical subperiosteal erosions and cysts of osteitis fibrosa cystica are present. Increased skeletal density has also been described.

The term PPH is used to indicate those patients whose physical appearance and roentgen features are those of PH, but whose blood chemistry levels are normal. The features of PH that result from derangement of metabolism of calcium and phosphorus are almost never present in PPH. Some of these features are cataracts, calcification of the basal ganglia and dentate nucleus, and abnormal tooth formation. PPH is presumed to represent a partial expression of the genetic abnormality. The complete form, manifesting all the major components of the disease, is represented by PH.

RESULTS

SKULL

The calvarium has been described as thick in approximately 37 per cent of the patients with PH and in 25 per cent of those with PPH (Table 1). It was present in 1 of our 3 patients (Fig. 1). Usually, this thickening was the result of widened diploe, although the inner table was described as thick in 3 patients with PPH and in 1 of PH.

Calcification of both the basal ganglia and the dentate nucleus of the brain has been reported in 44 per cent of the patients with PH. It was seen in only 1 of our 3, not appearing until the age of 17 years. This

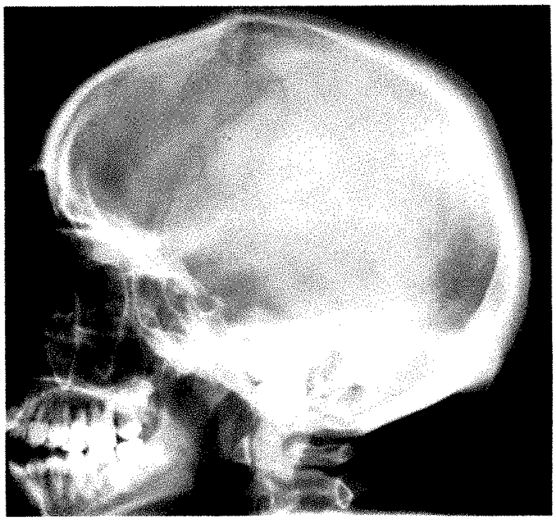

FIG. 1. The calvarium is moderately thickened.

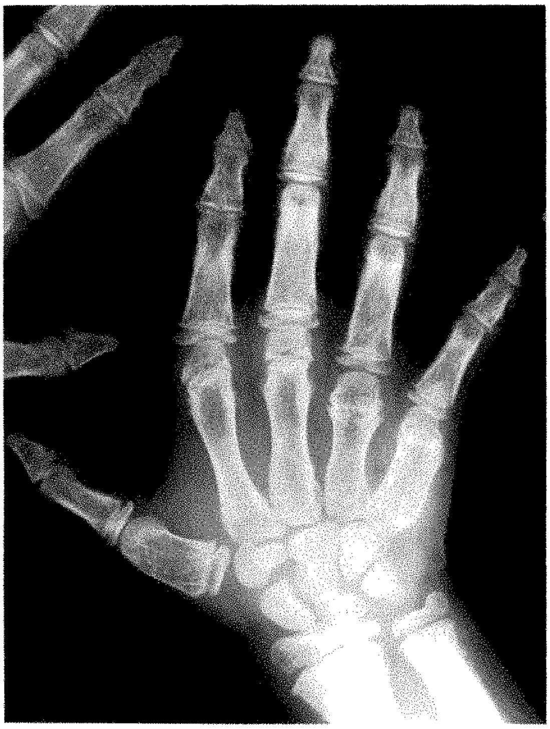

Fig. 2. Typical roentgen changes of pseudohypoparathyroidism. All of the tubular bones are short. The first, fourth, and fifth metacarpals are disproportionately short with diminished tubulation, and their epiphyses are partially fused. All of the distal phalanges and the second and fifth middle phalanges are extremely short. Extensive subperiosteal resorption, indicative of osteitis fibrosa resulting from secondary hyperparathyroidism is visible on all of the phalanges and on the radius and ulna. (Reproduced with permission from *J. Clin. Endocrinol. & Metabol.*[33])

calcification has been described in 1 patient with PPH, and also in 1 with PH whose calcium and phosphorus values reverted to normal spontaneously. Calcification of other parts of the brain was noted in 15 per cent of PH and 8 per cent of PPH patients. Calcification in the brain usually develops late in childhood or early adult life and is independent of other soft-tissue ossification or calcification. It is related to the abnormal serum concentration of calcium and phosphorus, whereas soft-tissue ossification in this syndrome appears to be genetic in origin. Calcification of the basal ganglia also occurs in idiopathic hypoparathyroidism.

Abnormal dentition (hypoplasia, defective dentine, widened root canals, delayed eruption, and excessive caries) was apparent in 37 per cent of PH patients. In 1 of our patients, eruption of teeth was delayed, many teeth were carious, and root canals were wide. As would be expected, this abnormal dentition was not mentioned in

Table II

ABNORMAL METACARPALS NOTED IN PUBLISHED CASE REPORTS OF BOTH PH AND PPH AND IN OUR PATIENTS WITH PH*

	PH				PPH	
	Published Case Reports (no.)	Our Patients (no.)	Total	Per Cent	Published Case Reports (no.)	Per Cent
Hands examined roentgenologically	102	6	108		40	
Normal	27	0	27	25	4	10
Abnormal	75	6	81	75	36	90
Abnormal, individual metacarpals not specified	18	0	18		2	
Abnormal, individual metacarpals specified	57	6	63		34	

* Data obtained from references: 1–3, 6–10, 13–15, 18–31, 34, 35, 37, 39, 42, 43, 45–51, 54–57, 59, 63, 66.

Table III

ABNORMAL PHALANGES OF HANDS NOTED IN PUBLISHED CASE REPORTS OF BOTH PH AND PPH AND IN OUR PATIENTS WITH PH*

	PH		PPH
	Published Case Reports (no.)	Our Patients (no.)	Published Case Reports (no.)
No. of patients with roentgen examinations of hands	51	3	20
No. of patients in whom abnormal phalanges were noted	26	3	15
No. of patients with these abnormal phalanges:			
Distal	17	3	12
Middle	16	1	10
Proximal	1	0	8
Not specified	1	0	0

* This table is unavoidably incomplete because of reasons discussed in the text. Data obtained from references: 1–3, 6–10, 13–15, 18–31, 34, 35, 37, 39, 42, 43, 45–51, 54–57, 59, 63, 66.

reports of patients with PPH whose blood calcium and phosphorus levels were normal.

HANDS

Abnormality of the skeletal structures consisted of disproportionate uniform shortening of metacarpals and phalanges (Fig. 2). This shortening involved both hands and tended to be symmetric, although occasionally involved different metacarpals and phalanges on the two sides. In patients with short stature, all of the bones, including those of the hands, were short but usually evoked no comment in the reports.

Tables II and III list the abnormal metacarpals and phalanges noted in the reviewed case reports and in our own patients. Table IV enumerates the individual abnormal metacarpals in these patients and Table V gives the individual abnormal phalanges in our patients with PH. In general, the reference to short digital bones means only disproportionate shortening. In all probability, most of the bones were shorter than normal when the patient was of short stature.

Metacarpals were short in about 75 per cent of the patients with PH and in 90 per cent of those with PPH. In PH the fourth and fifth metacarpals were nearly always involved with the first, third, and second next. In PPH, the order of frequency was in the fourth, fifth, third, first, and second metacarpals. The second metacarpal was short in only 27 per cent of the patients with PH and in 26 per cent with PPH. In our own 3 patients, the first, fourth, and fifth metacarpals were disproportionately

Table IV

INCIDENCE OF SHORT OR DEFORMED METACARPALS NOTED IN PUBLISHED CASE REPORTS OF BOTH PH AND PPH AND IN OUR PATIENTS WITH PH*

	PH				PPH	
	Published Case Reports (no.)	Our Patients (no.)	Total	Per Cent	Published Case Reports (no.)	Per Cent
Hands in which abnormal metacarpals were specified	57	6	63		34	
Metacarpal I abnormal	46	4	50	79	13	38
Metacarpal II abnormal	15	2	17	27	9	26
Metacarpal III abnormal	28	3	31	49	17	50
Metacarpal IV abnormal	53	4	57	91	29	86
Metacarpal V abnormal	54	4	58	92	26	77

* Data obtained from references: 1, 7, 8, 13–15, 18–20, 22–24, 26, 28, 29, 31, 34, 35, 37–39, 42, 43, 45–49, 54, 56, 57, 59.

TABLE V

ABNORMAL PHALANGES OF HANDS IN OUR PATIENTS WITH PH

No. of hands examined roentgenologically	6		
Normal phalanges	0		
Abnormal phalanges (short or deformed)	6		
Incidence of abnormal phalanges:	Distal	Middle	Proximal
Digit I	6	0	0
Digit II	6	2	0
Digit III	5	0	0
Digit IV	4	2	0
Digit V	4	0	0

short in 67 per cent, the third in 50 per cent, and the second in 33 per cent. Fusion of the epiphysis to the metaphysis was usually premature in disproportionately short metacarpals and phalanges, but the shortening had preceded fusion. In some short bones, fusion had occurred at the expected time, indicating a disturbance in proliferation of the epiphyseal growth cartilage.

Usually, short phalanges were not described, although they were sometimes visible in published reproductions of roentgenograms. Frequently, they were poorly reproduced or not included on roentgenograms. Nevertheless 26 instances of PH and 15 of PPH with at least one short phalanx were documented. All but 1 were associated with short metacarpals. Shortening was most frequent in the distal and middle phalanges. Although frequency was not tabulated, we gained the impression that shortening is most common in the distal first phalanx. Shortening involved the first and second distal phalanges in all 3 of our

TABLE VII

ABNORMAL PHALANGES OF FEET NOTED IN PUBLISHED CASE REPORTS OF BOTH PH AND PPH AND IN OUR PATIENTS WITH PH*

	PH		PPH
	Published Case Reports (no.)	Our Patients (no.)	Published Case Reports (no.)
No. of patients with roentgen examinations of feet	25	3	14
No. of patients in whom abnormal phalanges were noted	3	3	7
No. of patients with these abnormal phalanges:			
Distal	2	1	5
Middle	2	0	5
Proximal	3	3	6
Not specified	0	0	1

* This table is unavoidably incomplete because of reasons discussed in the text. Data obtained from references: 1–3, 7–9, 15, 18–20, 22, 24–26, 28–30, 36–39, 46, 47, 50, 51, 61, 66.

TABLE VI

ABNORMAL METATARSALS NOTED IN PUBLISHED CASE REPORTS OF BOTH PH AND PPH AND IN OUR PATIENTS WITH PH*

	PH				PPH	
	Published Case Reports (no.)	Our Patients (no.)	Total	Per Cent	Published Case Reports (no.)	Per Cent
No. of feet examined roentgenologically	50	6	56		28	
Normal	15	2	17	30	1	3
Abnormal (short or deformed)	35	4	39	70	27	97
Abnormal, individual metatarsals not specified	6	0	6		4	
Abnormal, individual metatarsals specified	29	4	33		23	

* Data obtained from references: 1–3, 7–9, 15, 18–20, 22, 24–26, 28–30, 36–39, 46, 47, 50, 51, 61, 66.

TABLE VIII

INCIDENCE OF SHORT OR DEFORMED METATARSALS NOTED IN PUBLISHED CASE REPORTS OF BOTH PH AND PPH AND IN OUR PATIENTS WITH PH*

	PH				PPH	
	Published Case Reports (no.)	Our Patients (no.)	Total	Per Cent	Published Case Reports (no.)	Per Cent
Feet in which abnormal metatarsals were specified	29	4	33		23	
Metatarsal I abnormal	9	2	11	33	12	52
Metatarsal II abnormal	6	0	6	18	6	26
Metatarsal III abnormal	19	2	21	64	14	61
Metatarsal IV abnormal	26	2	28	85	17	74
Metatarsal V abnormal	10	0	10	30	8	35

* Data obtained from references: 1, 7–9, 15, 18–20, 22, 24, 26, 29, 30, 37–39, 46, 47, 50, 61.

patients and the other distal phalanges were short in 72 per cent of the fingers. The middle phalanges of the second and fourth fingers of both hands were short in 1 of these patients and the proximal phalanges were not abnormal.

FEET

Tables VI and VII list the abnormal metatarsals and phalanges noted in the reviewed case reports and in our own patients. Table VIII enumerates the individual abnormal metatarsals in these patients and Table IX gives the individual abnormal phalanges in our patients with PH.

TABLE IX

ABNORMAL PHALANGES OF FEET IN OUR PATIENTS WITH PH

No. of feet examined roentgenologically	6			
Normal phalanges	0			
Abnormal phalanges (short or deformed)	6			
Incidence of abnormal phalanges:		Distal	Middle	Proximal
Digit I		2	0	6
Digit II		2	0	6
Digit III		2	0	6
Digit IV		2	0	6
Digit V		2	0	6

Of 50 feet studied in reproduced roentgenograms of patients with PH, approximately 70 per cent were abnormal. All of these had at least 1 short metatarsal with the fourth and third predominating (85 per cent and 64 per cent, respectively, [Fig. 3]). The first, second, and fifth were short in 33, 18, and 30 per cent, respectively. In roentgenograms of patients with PPH, all feet, except in 1 patient, were abnormal. The fourth (74 per cent) and third (61 per cent) metatarsals were most commonly involved. Next in order were the first (52 per cent), the fifth (35 per cent) and the second (26 per cent). In 2 of our 3 patients, the first, third, and fourth metatarsals were disproportionately short. The phalanges of the feet are more difficult to evaluate because of their common normal variations: short middle phalanges, which frequently lack epiphyses; absent fifth middle phalanx; and cone-shaped epiphyses of the proximal phalanges. The short middle phalanges may therefore be disregarded. Abnormal phalanges were noted only in 3 of 25 reports of PH and in 7 of 14 PPH. Short distal phalanges were described in 10 per cent of the patients with PH and 23 per cent of those with PPH. The proximal phalanges were short in 13 per cent and 27 per cent, respectively. The distal phalanges were short in 1

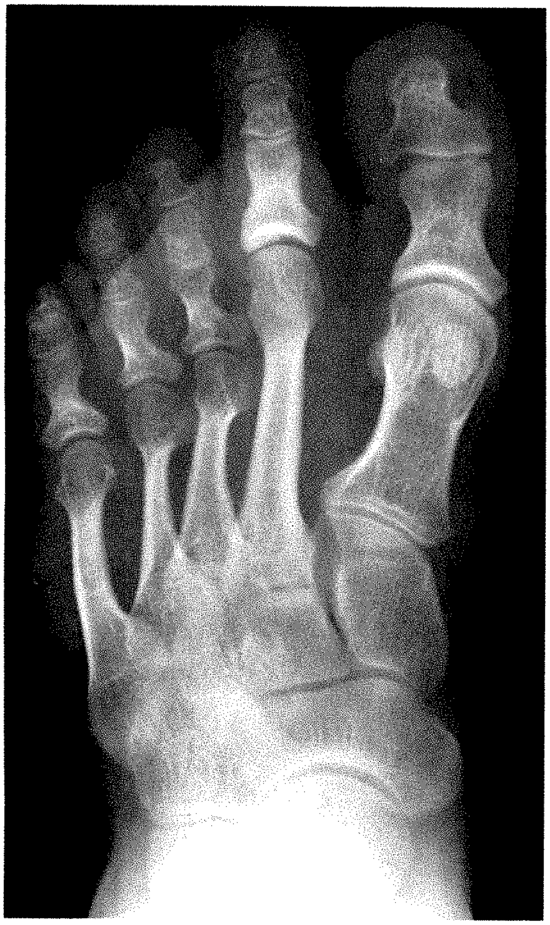

FIG. 3. Same patient as in Figure 1 and 2. The first, third, and fourth metatarsals are relatively shorter than the other short metatarsals. In contrast to the short metacarpals, the third and fourth short metatarsals are usually well tubulated.

of our patients and the proximal phalanges in all 3. Premature epiphyseal fusion of short bones was common in the feet of our patients, as well as in those described in the literature.

Among other skeletal anomalies, exostosis was reported in 2 patients with PH and in 5 with PPH. Exostosis was present in 2 of our 3 patients (Table x). These exostotic growths differ from the usual osteochondromas. Usually, they are not located in the metaphyseal region but are more central. They are frequently broadbased and short, and project at approximately right angles to the shaft. In 1 of our patients observed for a number of years, an exostosis began as a soft-tissue ossification at the proximal end of the femur and ultimately fused to the bone (Fig. 4, *A* and *B*). More than one such growth may be present in PH and PPH. They are not as extensive or as symmetric, however, as those in "multiple familial exostosis." Neither are they associated with localized bone deformities, as in "multiple familial exostosis."

HIPS

Coxa vara has been reported in 2 patients with PH and in 2 with PPH (Table x). In 1 of our 3 patients, bilateral coxa vara was preceded by a defect of ossification in the zone of provisional calcification. This ossification was similar to that seen in rickets, metaphyseal dysostoses, or congenital coxa vara. It also resembled the metaphyseal defects of the knees, as seen in genu valgum. It was limited, however, to the proximal femoral metaphysis and was succeeded by slipping of the epiphyses (Fig. 5). We have seen similar changes in 2 other patients who are not included in this report.

Coxa valga was reported in 3 patients with PH and in 1 patient with PPH, and in 1 of our 3 (Fig. 4, *A* and *B*).

A groove-like deformity of the proximal articular surface of the phalanges was reported in 3 patients with PPH. Other defects, which we call cone-shaped epiphyses, in the adjacent epiphyseal metaphyseal regions of the phalanges and metacarpals were reported in 4 patients with PH and were seen in 2 of our patients (Fig. 2).

Bowing of the long bones was described with PH in 5 patients.

Including our reports, accelerated maturation of the skeleton was reported in 9 patients with PH and in 1 patient with PPH. Usually, this maturation was inferred from premature epiphyseal fusion of the tubular bones of the hands and feet. Selective premature fusion occurred in the hands and feet of all 3 of our patients. A slight acceleration of maturation of other bones was also observed. In 2 of our 3 patients, 6 and 6½ years of age, this maturation was advanced 14 and 18 months, respectively.

Table X

MISCELLANEOUS ABNORMALITIES NOTED IN PUBLISHED CASE REPORTS OF BOTH PH AND PPH AND IN OUR PATIENTS WITH PH*

	PH				PPH	
	Published Case Reports (no.)	Our Patients (no.)	Total	Per Cent	Published Case Reports (no.)	Per Cent
Total cases	57	3	60		20	
Coxa vara	2	1	3	5	2	10
Coxa valga	3	1	4	7	1	5
Slipped capital femoral epiphyses	1	1	2	3	0	0
Anteroposterior grooves of proximal base of phalanges	2	0	2	3	3	15
Cone-shaped epiphyses of digits	4	2	6	10	0	0
Bowed long bones	5	0	5	8	0	0
Reported accelerated skeletal maturation	6	3	9	15	1	5
Exostoses	2	2	4	7	5	25
Soft tissue calcification or ossification	30	3	33	55	8	40
Osteoporosis	12	1	13	22	1	5
Abnormally dense skeleton	3	0	3	5	0	0
Prominent trabeculae	7	0	7	12	1	5
Osteitis fibrosa (hyperparathyroidism)	3	3	6	10	0	0

* Data obtained from references: 2–4, 6–10, 13–15, 18–28, 31, 33–39, 41–43, 46, 48–51, 54–57, 61, 63, 66.

OSTEOPOROSIS AND OSTEOSCLEROSIS

Osteoporosis was reported in 12 patients with PH and in 1 patient with PPH (Table x). One of our patients had bone with coarse trabecular pattern and diminished density, the cause of which is unknown. Of the reported patients with PH, 3 had osteosclerosis. This osteosclerosis is also seen occasionally in idiopathic hypoparathyroidism and is then presumably the result of diminished osteoclastic resorption secondary to decreased or absent parathyroid hormone. In PH, however, the parathyroids are intact and secrete a normal or excessive amount of hormone. Resorption of bone may be inadequate, however, if the hormone is abnormal or the bone is resistant to resorption.

OSTEITIS FIBROSA CYSTICA

We have previously reported 2 patients[58] with PH in whom roentgen evidence of osteitis fibrosa cystica consisted of typical subperiosteal resorption of the cortex. This resorption was particularly evident in the middle phalanges (Fig. 2). It responded subsequently to treatment, which restored the abnormal serum calcium and phosphorus levels to normal. We assumed that this resorption was caused by secondary hyperparathyroidism, the result of the low serum calcium. Since then, 3 additional patients have been reported[10,21,55] and we have seen a third patient. This totals 6 cases of PH (Table x). That secondary hyperparathyroidism is not more frequent in PH is surprising. In 1 of our patients, subperiosteal resorption was not seen initially but appeared at the age of 4 years.

SOFT-TISSUE OSSIFICATION OR CALCIFICATION

Ossification and calcification of soft tissue, exclusive of the basal ganglion, dentate nucleus, and other parts of the brain, are 2 of the common features of both PH and PPH. This change was reported in 33 (55 per cent) and 8 (40 per cent) patients, respectively. In many, it was probably

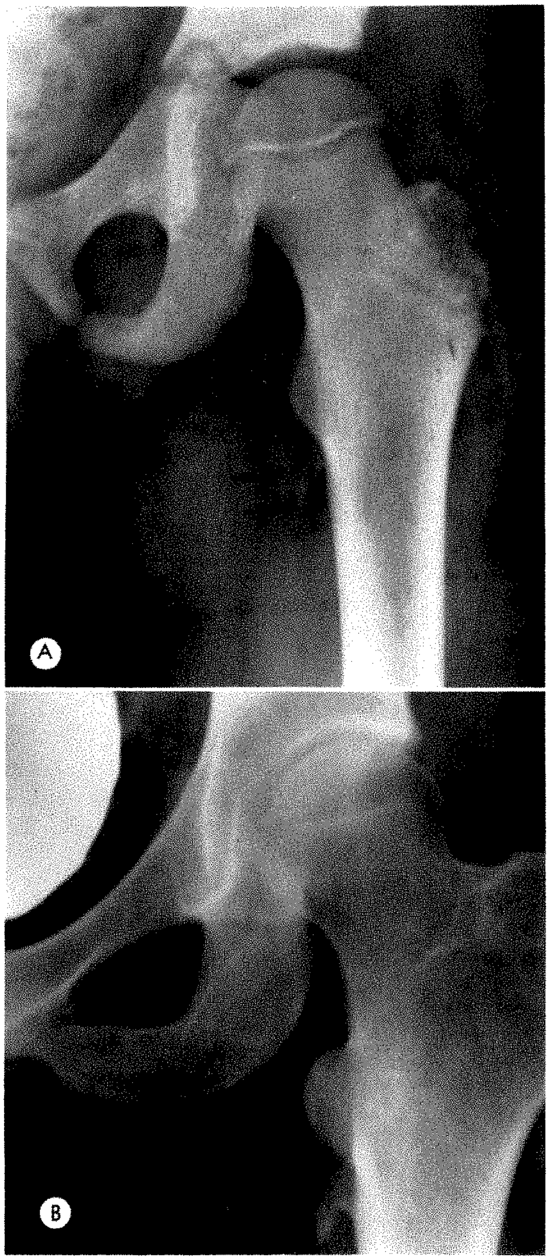

Fig. 4. (*A*) Ossification of the soft tissue adjacent to the medial femoral cortex. (*B*) The ossification has now attached to the cortex and appears to be arising from it. Another exostosis is attached to the pubic bone. Coxa valga is present.

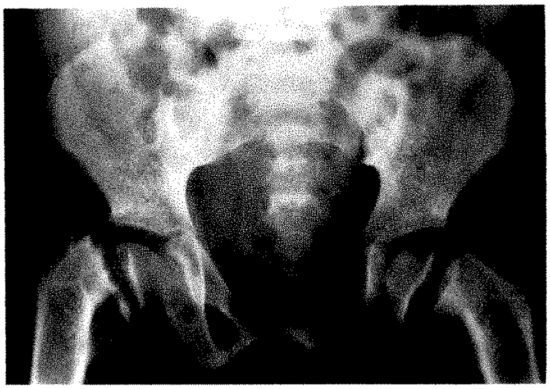

Fig. 5. Almost symmetric coxa vara deformities of both femurs. The neck is bent caudally and its proximal segment is not mineralized. Both epiphyses have slipped caudally. Similar lesions occur with rickets, metaphyseal dysostosis, primary secondary hyperparathyroidism and congenital coxa vara. The distal femur was normal. (Reproduced with permission from *J. Clin. Endocrinol. & Metabol.*[33])

undetected because total body roentgenograms are not ordinarily obtained and the mineral deposits are usually small and easily missed. In our 3 patients such densities were found in each (Fig. 6). The calcium or bone may be present at birth and, if superficial, may produce nodules which can be seen or palpated. These nodules involve connective tissue structures of the skin, subcutaneous tissue, fascial planes, or ligaments and tendons. Periarticular regions appear to be particularly favored. Both sides of the body may be involved,

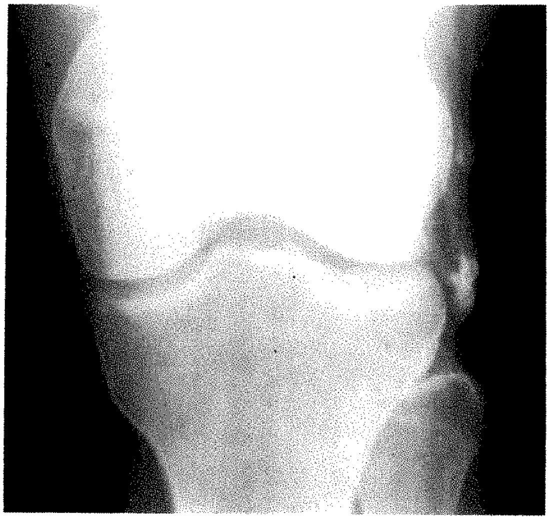

Fig. 6. Unusually large and dense ossification of soft tissue along the lateral surface of the knee. Frequently, ossification and calcification are barely discernible on roentgenograms.

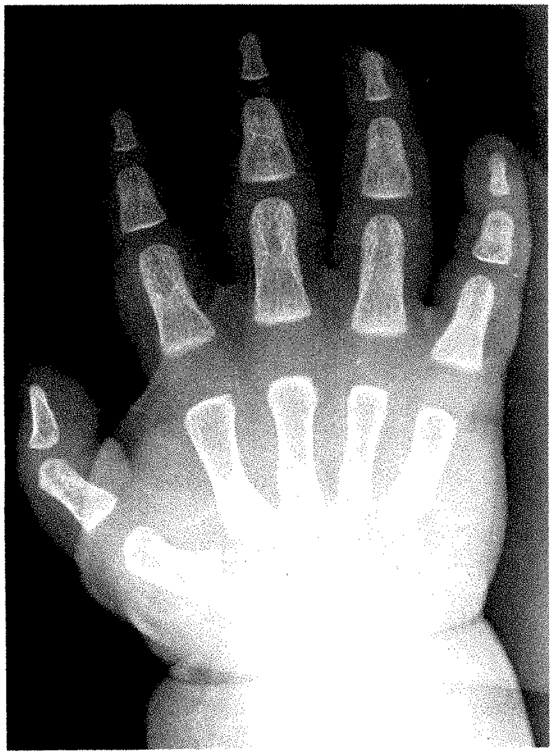

Fig. 7. The right hand at 9 months of age. The only abnormality is a decreased distance between the first distal and proximal phalanges. Subsequent roentgenograms showed a congenital absence of the distal epiphysis.

but not symmetrically. The nodules are common in the scalp and in the upper and lower extremities, particularly in the hands and feet. In some of our patients, the neck, trunk, and abdominal and thoracic walls have been involved as well. On roentgenograms, small punctate densities may be seen which in some regions coalesce. The densities remain the same size or increase slightly with advancing age. They may be or, perhaps always are, present before the characteristic bony abnormalities appear.[58] Since they are present in PPH with normal serum concentration of calcium and phosphorus, they are probably genetically determined. Also they are independent of basal ganglion and dentate nucleus calcification which is related to abnormal calcium and phosphorus metabolism. One of our patients with soft-tissue ossification at birth, initially had hypercalcemia, which subsequently reverted to hypocalcemia with hyperphosphatemia. The relationship of the hypercalcemia to ossification of soft tissue is not known. Hypercalcemia in PH had not been reported previously.[58] The calcification or ossification does not involve the muscles and viscera.

DISCUSSION

The full syndrome of PH is characterized by 3 basic genetically determined components. Any one of these constitutents may be only partially manifested or may be absent. They are:

(1) Skeletal deviations consisting of short stature, disproportionate length of the digits of hands and feet, and thick calvarium. Other skeletal abnormalities which may be genetic have also been present occasionally. Some of these are exostosis, coxa vara, coxa valga, bowed radius and ulna, and dense or lucent bones.

(2) Ossification or calcification of soft tissues, excluding those in the basal ganglion or dentate nucleus of the brain. This ossification or calcification differs from that seen in other syndromes. It is present early in life, is usually not extensive, and increases slightly with growth of the patient. It involves the skin, subcutaneous tissues, and deeper connective tissue, but not muscle, vessels, cartilage, or viscera. These features tend to differentiate this ossification or calcification from calcinosis universalis, dermatomyositis, and myositis ossificans. The accumulation of bone or calcium may be associated with or without abnormalities of calcium and phosphorus metabolism, of which they are independent. Such soft-tissue calcification and ossification, because of its apparent genetic basis, should not occur in idiopathic or postoperative hypoparathyroidism. Nevertheless, it has been reported in a few isolated cases.

(3) Abnormally low serum concentration of calcium and high serum concentration of phosphorus, similar to those of hypoparathyroidism but resistant to injected parathyroid hormone. Possibly, this resistance results from reduced sensitivity of the

kidney to parathyroid hormone, diminishing phosphate diuresis and elevating serum of phosphorus. Secondary effects of the low calcium and high phosphorus are calcification of basal ganglion, faulty dentition, cataracts, and osteitis fibrosa cystica. Calcification of the basal ganglion also occurs in idiopathic or postoperative hypoparathyroidism. It has been seen rarely in patients with PPH who probably originally had PH with subsequent remission of the metabolic disturbance. This calcification is also present in other conditions, such as toxoplasmosis or idiopathic familial calcification of the basal ganglia.[11] The defects of the teeth consist of delayed eruption, faulty root formation, wide root canals, impaired formation of dentine, and caries.

The low blood calcium stimulates the parathyroid glands. The glands then elaborate excessive quantities of parathyroid hormone which acts on bone to produce osteitis fibrosa cystica. This latter manifestation is not as common as expected. As in other forms of secondary or primary hyperparathyroidism, however, bone lesions may not be visible on roentgenograms. Subperiosteal resorption, indicative of osteitis fibrosa cystica, was visible on roentgenograms of all 3 of our patients with PH. Thus, patients with PH have been described whose metabolism was defective but skeletal development was normal. These patients were with or without ectopic ossification or calcification. In others, metabolic defects and skeletal abnormalities were present but the soft tissues were normal.

In patients with typical skeletal abnormalities, with or without soft-tissue ossification or calcification but who respond normally to parathyroid hormone, the disease is called PPH. The following evidence suggests that PPH is an incompletely expressed form of a genetically determined abnormality of which PH represents the complete syndrome: (1) the sex distribution (almost twice as many females as males) is the same in the two abnormalities; (2) PPH has occurred among some relatives of patients with PH; (3) hypocalcemia may be an inconstant feature in an individual case which thus may be classified as one or the other at different times;[37,44] (4) the distribution and extent of genetically determined lesions are similar (excluding those due to hypercalcemia such as basal ganglion and dentate nucleus calcification, abnormal dentine formation, and cataracts).

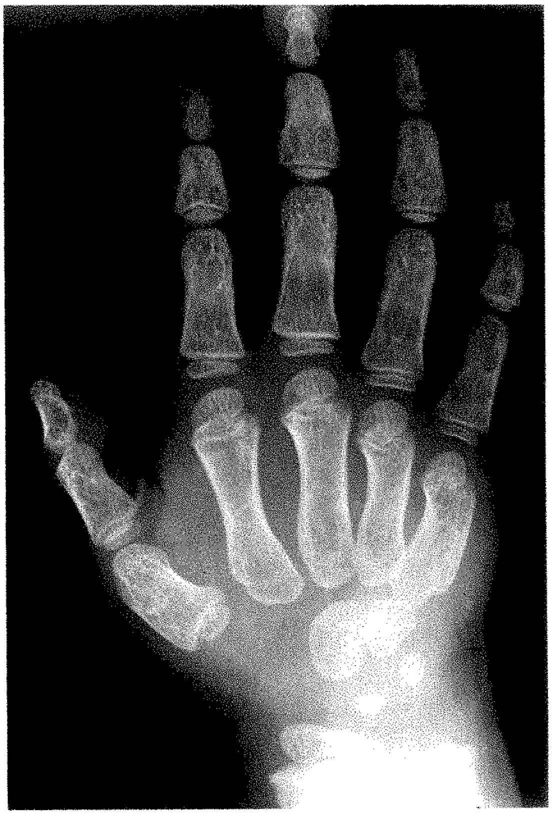

Fig. 8. Right hand at 49 months of age. All of the metacarpal bones are short. The cartilaginous growth plate of the first and fifth metacarpals is narrow, indicating that fusion is likely to take place prematurely. Almost complete lack of diaphyseal constriction of these 2 bones and, to a lesser extent, of the middle metacarpal is demonstrated. The phalanges are short and broad. The epiphyses of the first distal phalanx are congenitally absent and those of the right second and third distal phalanges are fused. The epiphyses of the fifth middle phalanx are in the process of fusion along the lateral margin. The epiphysis of the second middle phalanx is cone-shaped, fitting into a corresponding deformity of the metaphysis. This deformity is frequently seen in pseudohypoparathyroidism and pseudo-pseudohypoparathyroidism. (Reproduced with permission from *Radiology*.[58])

The most obvious changes roentgenologically are the short metacarpals, phalanges, and metatarsals. Usually, all of the tubular bones of the hands and feet are short, but some are shorter than others. The metacarpals most commonly and severely involved are the fourth and fifth with the first and third next. The distal and middle phalanges of the hand are more often short than are the proximal phalanges. In addition to their shortening, the metacarpals are usually broad because of diminished constriction of the center of the shafts. This diminished constriction is proportionate to the degree of shortening. The metaphyseal margins adjacent to epiphyses are flared. The epiphyseal growth cartilage is narrow and the epiphyses frequently fuse early.

Although the metatarsals are short and their epiphyses fuse early, except for the first, they are usually not excessively broad. Abnormal shortening and premature fusion of the phalanges, metacarpals, and metatarsals are frequently associated with cone-shaped deformity of the epiphyses and metaphyses. The fusion occurs first in the apex of the cone and then extends to the periphery (Fig. 2). In some phalanges the deformity is limited to the metaphyses. Some phalanges lack epiphyses or have rudimentary epiphyses, which are partially fused when they first appear.

We have reported a patient[68] with PH in whom typical skeletal abnormalities, not present initially, developed between 9 and 49 months of age (Fig. 7 and 8).

DIFFERENTIAL DIAGNOSIS

BRACHYPHALANGIA

Short metacarpals and metatarsals, similar in all respects to those seen in PH and PPH, may occur as isolated defects. They may also be associated with malsegmentation, or with errors in growth of the phalanges. These short fingers may be included under the term of brachyphalangia, which has been further categorized depending upon the specific bones involved. The deformities of the hands and feet are frequently hereditary and, in many instances, are associated with short stature. They are usually transmitted as a simple dominant characteristic. The bones involved are frequently bizarre in appearance and relatively symmetric on the 2 sides of the body. The usual involvement of the fourth and fifth and sometimes the first metacarpal, with relatively normal second metacarpal in PH and PPH, usually does not prevail. None of the patients with brachyphalangia has ectopic bone or calcium deposits. Aberrations of calcium and phosphorus metabolism do not develop in them or in members of their families.[58]

PERIPHERAL DYSOSTOSIS

Peripheral dysostosis is a dysplasia of bone in which the major manifestations are shortening of the metacarpals, metatarsals, and phalanges (Fig. 9, *A* and *B*). The short metacarpals are underconstricted and undertubulated with associated flaring of the metaphyses. The short phalanges are broad and frequently have cone-shaped epiphyses and metaphyses, most often present in the proximal phalanges. The epiphyses may fuse prematurely or may be congenitally absent. The styloid process of the ulna may be exceptionally long or may be retarded in development. This description would fit some of those of PH or PPH, but differs in the following features: (a) ossification or calcification of soft tissues is absent; (b) the phalanges and metacarpals, although short, are usually proportionate, whereas in PH and PPH they are usually disproportionate; (c) the cranial vault is not thick; and (d) the serum calcium and phosphorus levels are always normal.

CHONDRO-ECTODERMAL DYSPLASIA

The chondrodysplasia of the hands and feet may resemble that of peripheral dysostosis, but polydactyly and fusion of carpal bones are frequent complications. The tubular bones of the hands and feet may be disproportionately short and the distal phalangeal epiphyses delayed in

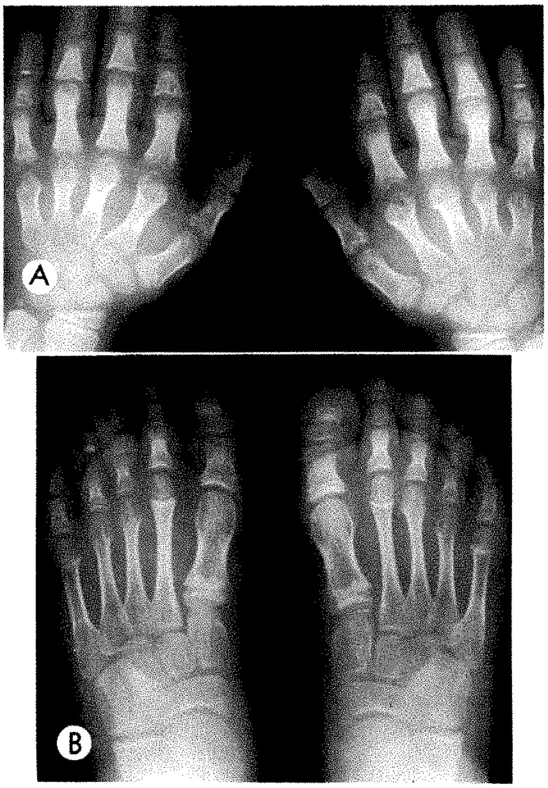

FIG. 9. Peripheral dysostosis. (*A*) All of the tubular bones are short. The proximal bones are relatively shorter with the distal phalanges least involved. The metaphyses of several phalanges are concave, some with adjacent cone-shaped epiphyses in various stages of fusion. This same change is also true of the first metacarpals. The articular margins of several epiphyses are notched. Epiphyses of the middle phalanges are either rudimentary or absent. (*B*) Similar changes are present in the feet.

maturation. The long bones of the extremities are shorter than normal, particularly, those distal to the elbows and knees. Ectodermal dysplasia is manifested as a dystrophy of the fingernails, sparse hair of the scalp, and delayed defective dentition.

With achondroplasia, Hurler's syndrome, Morquio's disease, congenital spondyloepiphyseal dysplasia, pseudoachondroplasia, and diastrophic dwarfism (Fig. 10), some or all metacarpals, metatarsals, and phalanges are short. They are easily distinguished, however, on the basis of the characteristic roentgen pattern.

Two diseases commonly produce lesions of the tubular bones of the hands and feet similar to those of PH and PPH. They are multiple familial exostosis and Turner's syndrome. Other features of these diseases readily distinguish them. Why these 2 syndromes and not those previously described are commonly included in the entity of PH or PPH is difficult to understand. All have in common the short metacarpals, metatarsals, and diminished stature.

MULTIPLE FAMILIAL EXOSTOSIS

This is a relatively common anomaly characterized by multiple osteochondromas or exostotic lesions in any of the bones except the cranial vault. In the long bones

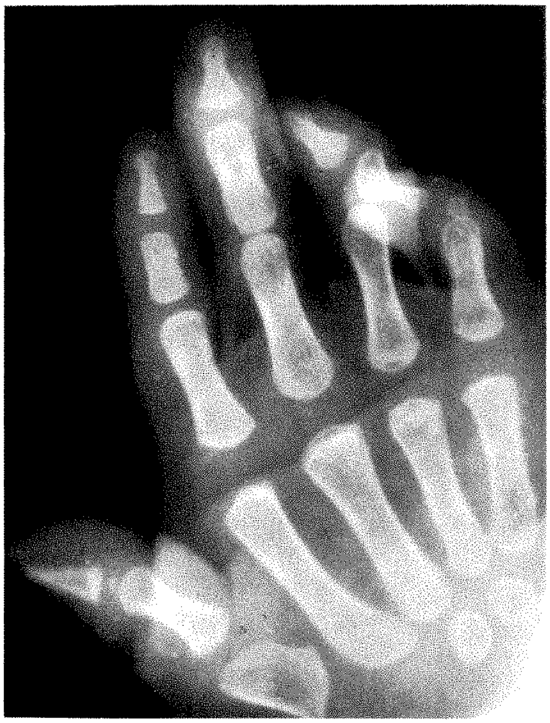

FIG. 10. Diastrophic dwarf. The striking abnormality of the hands in this disease is an extremely short first metacarpal, frequently triangular in shape. Other more variable errors of segmentation occur, such as the extremely short fourth and fifth phalanges and moderately short middle phalanx as in this patient. The epiphysis for the third middle phalanx is fused to the metaphysis. These patients also have scoliosis, clubbed feet, short tubular bones, multiple dislocations, and delayed appearance and deformity of many epiphyses.

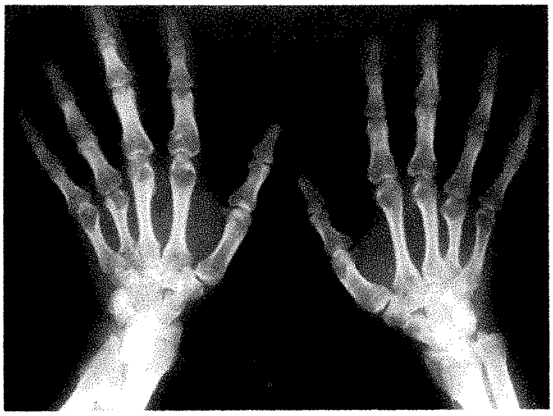

Fig. 11. Multiple familial exostosis. Bilateral short fourth metacarpal and the right first metacarpal resemble those seen in pseudohypoparathyroidism and pseudo-pseudohypoparathyroidism. The distinguishing features are the exostoses of several tubular bones which are usually present, the normal distal phalanges, and the characteristic shortening and deformity of the left ulna.

they are predominantly in the metaphyseal region, which is frequently widened. They usually point toward the middle of the shaft but may extend at right angles to the cortex. These lesions may be short or long, broadbased or pedunculated, or rough or smooth. In children, and sometimes in adults, a cartilage cap may be calcified. The exostotic growths are small in children and grow along with other bones of the skeleton. Their growth is accelerated during the prepubertal growth spurt. It is during puberty frequently that they are palpated and the diagnosis is established. After puberty the exostosis may remain stationary or continue at a slower rate. Many of these patients are short in stature, have short metacarpals, metatarsals, and phalanges, and curved radii or ulnae (Fig. 11). All of these anomalies are present with PH and PPH. The exostotic lesions in our patients with PH and PPH, and many of those illustrated, however, are not identical to those of multiple familial exostosis. In PH and PPH, exostosis tends to be located near the center of the shaft and extends at right angles to the cortex. In PH and PPH, it appears to rise from periosteal proliferation rather than from endochondral ossification. The growths are not associated with widening and distortion of the ends of the bone. Although multiple, they are not as numerous as in most cases of multiple familial exostosis. In 1 of our patients, an exostotic growth arose in the soft tissue adjacent to the proximal femur. It increased in size, ultimately fusing with the cortex (Fig. 4, *A* and *B*).

Usually, with multiple familial exostosis, one or both distal ulnae are short, broad, and irregular in contour. These abnormalities are unique to that disease. Irregularity in contour of the phalanges and periosteal or central enchondromas are sometimes present (Fig. 11). In these patients none of the other characteristic roentgen or chemical manifestations of PH or PPH is noted. The mode of genetic transmission in PH and PPH is probably as a sex-linked dominant with predominance in the female (2:1). It is probably not transmitted from father to son[37] although a few reports to the contrary have appeared.[17,36,40] In multiple familial exostosis, the transmission is autosomal dominant, predominating in the male with a ratio of 7:3. Transmission is usually from the father.[32]

TURNER'S SYNDROME

This syndrome consists of gonadal aplasia, short stature, webbed neck, and an increased carrying angle of the arms. It is usually accompanied by alterations in size, composition, or contour of skeletal components. Among these are a relatively short fourth metacarpal and an increased angle of the proximal carpal bones. The ulna may be short or the ulnar styloid process absent. The radius and ulna may be curved and the medial tibial condyle deformed, with a beak-like process arising from the medial metaphysis. A large medial femoral condyle, osteoporosis, and, at puberty, a delayed skeletal maturation are other possible alterations. The short fourth metacarpal is usually bilateral and of normal contour. It is often so slight that it can be detected only by comparing its length with the adjacent metacarpals. This comparison is made by drawing a line tangential to the distal end of the fifth and fourth metacarpals. The line should pass distal to the head of the third metacarpal.[5] In 1 of the 17 patients (6 per cent) with gonadal dysgenesis described by Archibald, Finby, and DeVito,[5] the fifth metacarpal was extremely short. It resembled the metacarpals in PH and PPH. Van der Werff ten Bosch[65] noted extremely short metacarpals with gonadal dysgenesis in 4 instances. He included these with 4 of PH in a syndrome which he called brachymetacarpal dwarfism. We have seen extremely short metacarpals in about 5 per cent of patients with Turner's syndrome. In these patients the metacarpals resembled those seen in PH and PPH (Fig. 12 *A*).

The lack of tubulation and constriction in all syndromes considered in this discussion is roughly proportional to the degree of shortening. This lack results in extremely short metacarpals that are relatively broad with widened metaphyses. They also have in common a narrow epiphyseal cartilage and concave metaphysis, with the epiphysis surrounded by the base of the metaphysis. The deformity of the metatarsals in PH and PPH is identical to that of Turner's syndrome (Fig. 12 *B*). Premature fusion of the epiphyses occurs in Turner's syndrome despite the retarded skeletal maturation. Short phalanges appear to be much more common in PH and PPH than in Turner's syndrome.

Turner's synrome and PH have many abnormalities in common. These are, short stature, curved long bones, short fourth or fifth metacarpals, and short metatarsals. Many more dissimilarities are present, however, which indicate that the 2 conditions are separate entities and should not be classified together.

Ossification or calcification of the soft tissues is not a feature of Turner's syndrome. Turner's syndrome has not been described in patients with metabolic disturbances of calcium and phosphorus. The cranial vault is not thick. The characteristic deformities about the knees of patients with Turner's syndrome are not seen with PH or PPH.

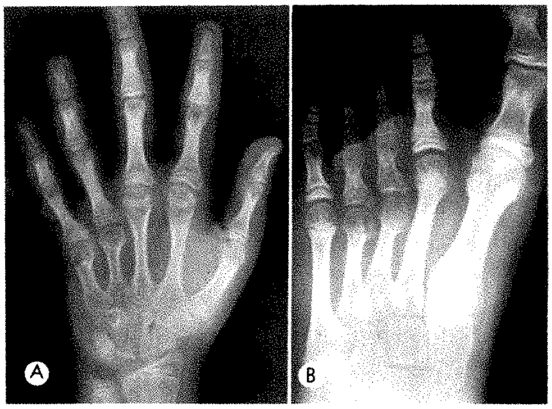

Fig. 12. Turner's syndrome. (*A*) Left hand. Short fourth and fifth metacarpals with premature fusion of the epiphyses and relative shortness of the fifth middle phalanx. The distal phalanges are normal. Mild osteoporosis is present. (*B*) Feet of a different patient with Turner's syndrome. Short third and fourth metatarsals with premature fusion of the epiphyses are the most striking deformities. Cone-shaped epiphyses of the middle three proximal phalanges are present but these, as well as the short middle phalanges and absent epiphyses of the third and fourth distal phalanges, occur frequently in otherwise normal individuals.

In Turner's syndrome, definite abnormalities of sex chromosomes occur, nearly always present in only 1 generation. This change differs from PH, in which the chromosome karyotype is normal and the disease is present in many generations.

SUMMARY

Pseudohypoparathyroidism (PH) is a hereditary congenital abnormality consisting of 3 separate components: (1) metabolic abnormalities of low serum calcium and high phosphorus which do not respond adequately to parathyroid hormone; (2) short stature and disproportionate shortening of some metacarpals, metatarsals, and phalanges, which may develop after birth; (3) ossification or calcification of the soft tissues. The soft tissue ossification or calcification may be the initial lesion with skeletal deformity and characteristic electrolyte disturbance developing later.

Pseudo-pseudohypoparathyroidism (PPH) is a partial manifestation of the same syndrome with normal calcium and phosphorus levels in the serum.

The skeletal and soft tissue lessions have been tabulated and are presented. Similar lesions of the metacarpals, metatarsals and phalanges occur with multiple familial exostoses, Turner's syndrome, and other less common syndromes, but these have other roentgen, clinical, and genetic findings that indicate the correct diagnosis.

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PROGRESSIVE SYNOSTEOSIS IN APERT'S SYNDROME (ACROCEPHALOSYNDACTYLY)*

WITH A DESCRIPTION OF ROENTGENOGRAPHIC CHANGES IN THE FEET

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ACROCEPHALOSYNDACTYLY was first suggested as a separate disease entity by Apert[1] in 1906. The classic description of this syndrome includes patients with a combination of acrocephaly and syndactylism. Since Apert's original description, there have been fewer than 200 cases reported.

The cause of the disease is attributed by Blank[2] to a single gene in heterozygous form, with sporadic cases resulting from new mutation in the germ cells in one or the other parent. An increased father's age appears to be an important causal factor.

Roentgenographic findings such as acrocephaly, bone fusions between the phalanges of different digits of the hands, and deformities of the shoulders and elbows have been described in the past, and will not be discussed in this paper. The synosteoses that occur with increasing age have not been appreciated in Apert's syndrome. This is due to the fact that most of the cases reported have been in young individuals, where serial studies at different ages have not been obtained. It is the purpose of the authors to describe and discuss the bone fusions that occur with increasing age, including roentgenographic changes in the feet.

The roentgenograms and clinical findings of the 9 patients under consideration in this article will be discussed in more detail in a separate publication. The ages of the patients at the time of their most recent roentgenograms are: Case 1, age 6 months; Case 2, age 2 years 2 months (hands), 4 years 7 months (feet); Case 3, age 3 years 9 months; Case 4, 3 years 10 months; Case 5, 5 years 6 months; Case 6, 14 years 10 months; Case 7, 26 years 6 months; Case 8, 54 years; Case 9, 67 years.

The roentgenographic findings in these patients are described under the headings: Spine, Hands and Feet.

SPINE

The synosteoses of the spine observed in this series of patients are limited to the cervical region. Fusions of the bodies of the cervical vertebrae are present in 2 cases. Roentgenograms obtained at the age of 8 years 6 months in Case 7 show discrete 6th and 7th cervical vertebrae, while later studies at the age of 26 years show fusion of these vertebrae (Fig. 1, *A* and *B*). Case 9, which shows fusion of the bodies of C-5, C-6, and C-7, does not have an earlier examination for comparison.

In Case 4, at the age of 3 years 10 months, there is fusion at the proximal end of the cervical spinous processes of C-2 and C-3 and between the entire length of the spinous processes of C-5 and C-6, which is not present at the age of 3 months (Fig. 2, *A* and *B*). A roentgenogram from Case 6, obtained at the age of 14 years, shows fusion of the cervical spinous processes at several levels, while an earlier study obtained shortly after birth shows no evidence of fusion of these processes. Case 5, in which there is fusion of the cervical spinous processes between C-2 and C-3 and between C-4 and C-5, does not have a prior examination for comparison.

HANDS

Six of the 9 patients show fusion between the 4th and 5th metacarpals. In Case 3, shortly after birth, there are separate 4th

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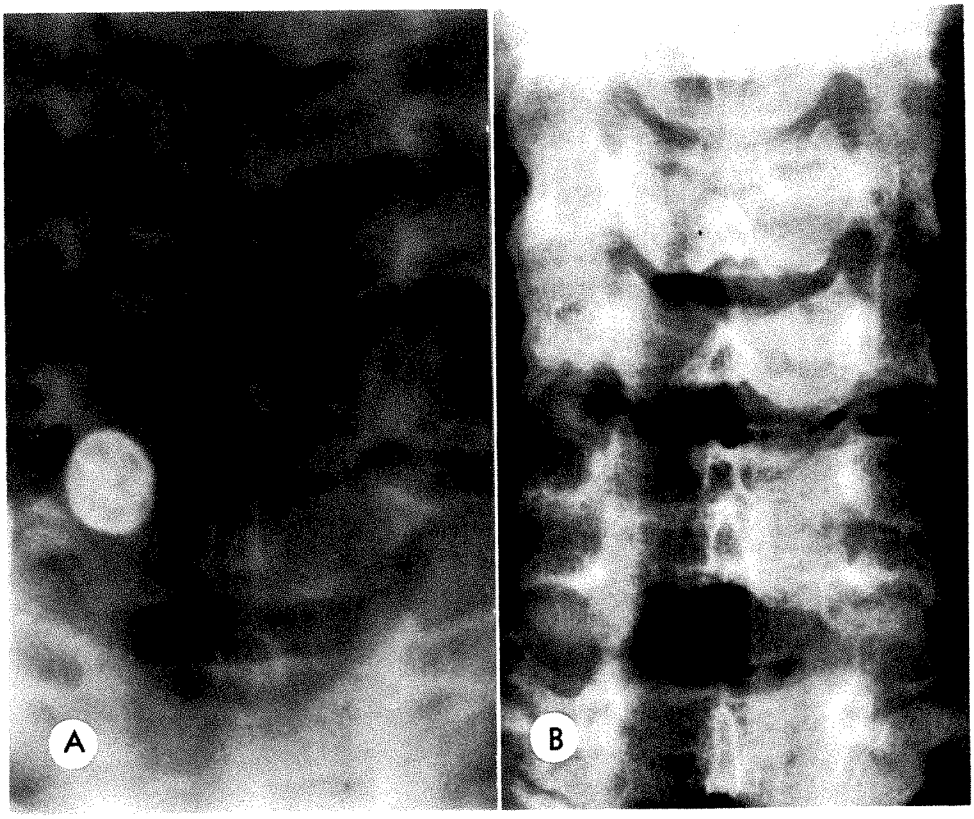

FIG. 1. *Case 7.* (*A*) At age 8 years 6 months, the 6th and 7th cervical vertebrae are discrete. (*B*) At age 26 years, there is fusion of the bodies of the 6th and 7th cervical vertebrae.

and 5th metacarpals, while at the age of 3 years 9 months there is fusion present between the proximal ends of these metacarpals. In Case 6, at the age of 6 years 6 months the 4th and 5th metacarpals are discrete, and at the age of 14 years 10 months fusion is present (Fig. 3, *A* and *B*). Case 6 also exhibits a Madelung's deformity and fusion between the phalanges of adjacent digits. Case 7 shows no evidence of fusion between the 4th and 5th metacarpals at the age of 8 years 6 months, while it is present at the age of 23 years. In Cases 3, 6, and 7, the ages at the time of the synosteoses are not known since roentgenograms were not obtained in the time intervals between the patients' ages listed above. Case 5, at birth, has fusion of the 4th and 5th metacarpals. Cases 1, 2, and 4, at the ages of 6 months, 2 years 2 months, and 3 years 10 months, respecspectively, do not have synosteosis between the 4th and 5th metacarpals. This represents an age range where fusion is not always observed so that the possibility of its occurrence at a later date, in these cases, is not excluded.

FEET

Fusion between the tarsal bones is observed to be one of the most constant findings in our cases of Apert's syndrome, and yet, in the majority of articles on this subject, this abnormality is not described. The infrequency with which this finding had been observed in Apert's syndrome is illustrated by the review of the world literature by Park and Powers[8] in 1920, where in 29 cases only 1 with tarsal fusion was noted. Two of the later exceptions are the papers by Cohn and Cooper. Cohn,[3] reporting on a single case, observed fusion of the tarsal bones, and Cooper,[4] in describing her patient, noted the tarsal bones "to

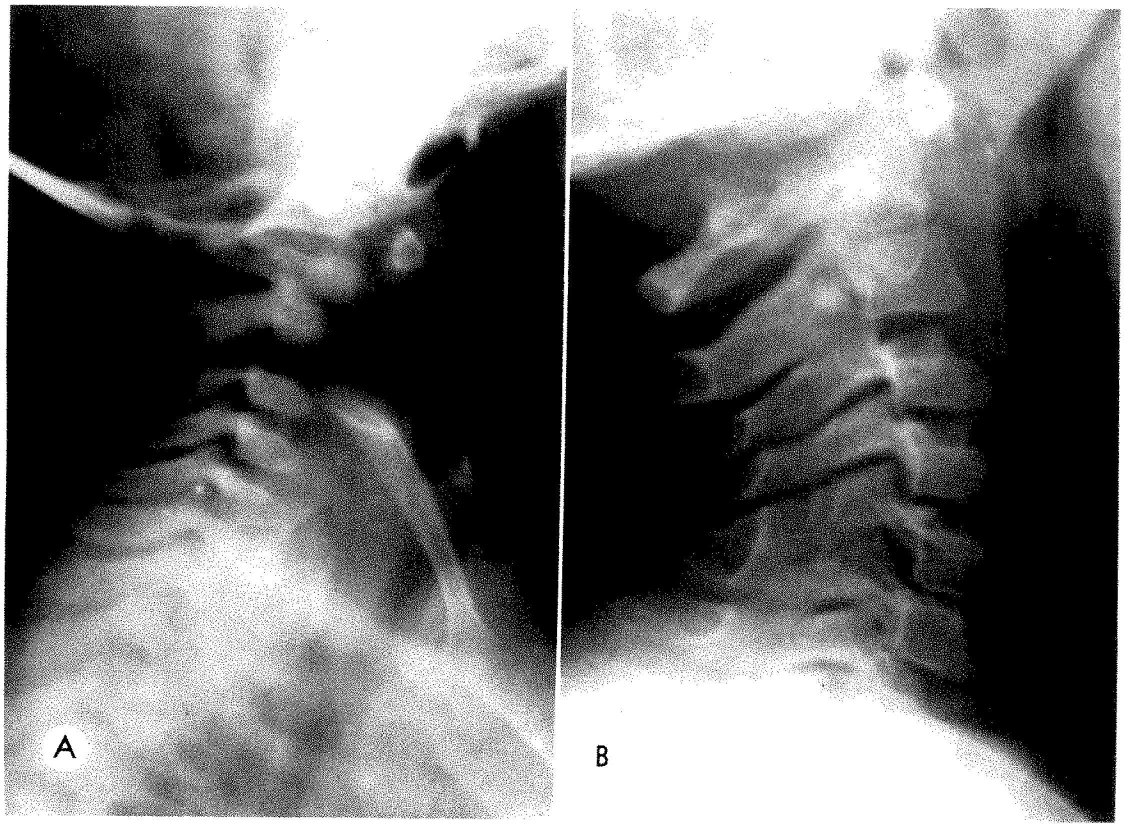

Fig. 2. *Case 4.* (*A*) At age 3 months, the cervical vertebrae have separate spinous processes. (*B*) At age 3 years 10 months, there is fusion of the spinous processes between C-2 and C-3 and between C-5 and C-6.

be irregularly fused and reduced in number." These authors do not mention the absence of fusion on earlier roentgenograms of their patients.

Progressive synosteosis of the tarsal bones is demonstrated by the following cases. Case 2 shows a discrete calcaneus and cuboid at the age of 5 months, followed by complete fusion of these bones at the age of 4 years 7 months (Fig. 4, *A* and *B*). In Case 5, the calcaneus and cuboid are separate at birth, and almost completely fused at the age of 2 years 6 months (Fig. 4*C*). The same case, at the age of 5 years 6 months (Fig. 4*D*), shows complete fusion of the calcaneus with the cuboid, and, in addition, fusion of the 3rd metatarsal with the lateral cuneiform. In Case 6, at the age of 11 years 6 months, the navicular and medial cuneiform are separate, while at the age of 14 years 10 months they are fused (Fig. 4, *E* and *F*).

Cases 7, 8, and 9 have tarsal fusion, but roentgenograms obtained during childhood are not available. With the progression of fusion shown in Cases 2, 5, and 6 and the synosteoses already established in the older

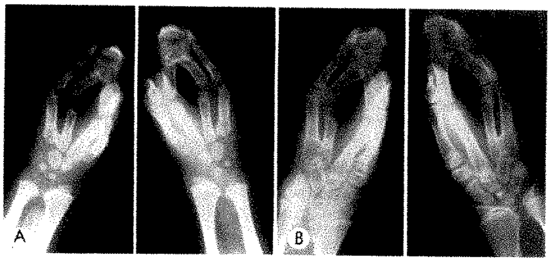

Fig. 3. *Case 6.* (*A*) At age 6 years 6 months, the 4th and 5th metacarpals are separate. (*B*) Eight years later there is bilateral fusion between the bases of the 4th and 5th metacarpals. Surgical changes are present with only the bases of the 2nd metacarpals remaining, and these are not well shown because of superimposition of adjacent metacarpals. Synosteosis between adjacent phalanges and Madelung's deformity are also demonstrated.

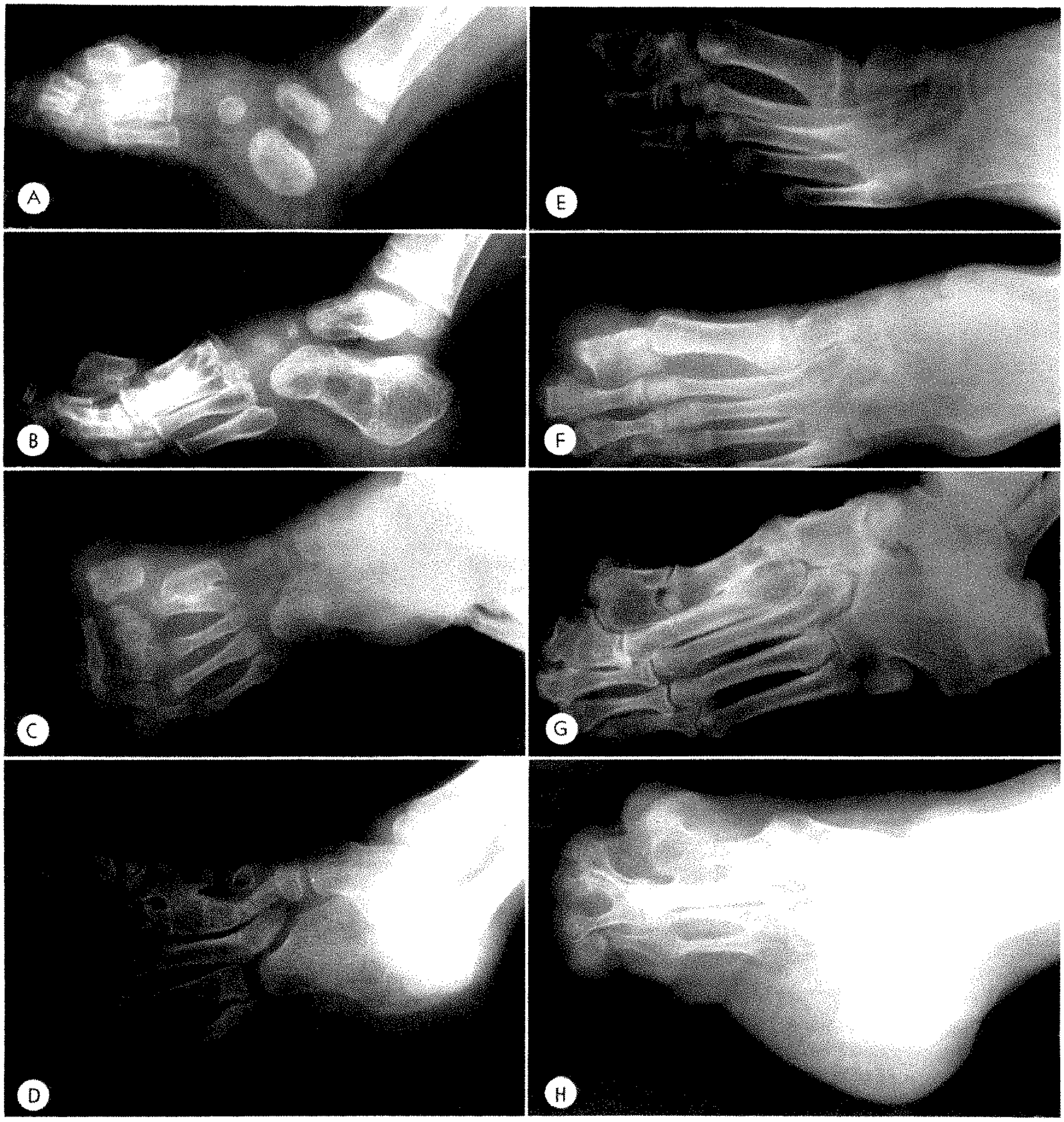

Fig. 4. (*A*) *Case 2*, age 5 months, with calcaneus and cuboid separate. (*B*) Case 2, age 4 years 7 months, with fusion of the calcaneus and cuboid.

(*C*) *Case 5*, age 2 years 6 months, with partial fusion of the calcaneus and cuboid, fusion between the 1st and 2nd metatarsals, and fusion between the terminal phalanges. (*D*) Case 5, age 5 years 6 months, with postsurgical changes, complete fusion of the calcaneus and cuboid, and fusion of the 3rd metatarsal and the lateral cuneiform.

(*E*) *Case 6*, age 11 years 6 months, with medial cuneiform and navicular separate. (*F*) Case 6, age 14 years 10 months, with fusion between the navicular and the medial cuneiform.

(*G*) *Case 8*, age 54, with fusions of: navicular and medial cuneiform, calcaneus and cuboid, navicular and calcaneus, 3rd metatarsal and lateral cuneiform, 1st and 2nd metatarsals, and between the interphalangeal joints. A "pseudo-articulation" between the phalangeal masses of the 1st and 2nd digits and a well developed os peroneum are also present. (*H*) *Case 9*, age 67, showing most of the changes seen in *G* and, in addition, fusion between the 4th and 5th metatarsals and the bases of the phalangeal masses of the 3rd and 4th toes.

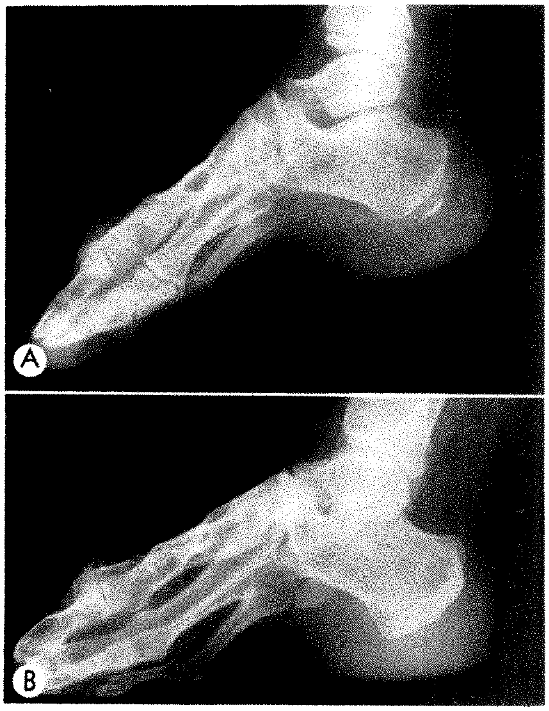

Fig. 6. *Case 7.* Left foot. (*A*) Roentgenogram at age 8 years. (*B*) Age 26 years 6 months, with fusion of the metatarso-phalangeal joints of the 3rd and 4th digits, development of a "pseudo-articulation" between the 2nd and 3rd metatarsals, and an os peroneum present at the base of the 5th metatarsal. None of these findings are seen in *A*.

patients, a definite pattern in the order of fusion at the tarsal bones becomes manifest. In Cases, 1, 2, and 5, with roentegnograms taken prior to 1 year of age, the calcaneus and cuboid are discrete (Fig. 4*A*). Partial to complete fusion of the calcaneus with the cuboid is seen in all patients older than 2 years 6 months of age (Fig. 4*B* and 6*B*). Fusion of the lateral cuneiform with the 3rd metatarsal is not present in Cases 1, 2, 3, and 4, but is present in the remaining cases, beginning with Case 5 at the age of 5 years 6 months (Fig. 4, *D*, *G* and *H*; and 5*B*). The next bone fusion in the foot appearing with increasing age (Case 7, age 8 years; Case 6, age 14 years 10 months) involves the navicular and medial cuneiform and usually also the intermediate cuneiform (Fig. 4, *F–H;* and 5, *A* and *B*). Cases 7 and 8 show bone fusion between the navicular and calcaneus on one side only, which with the synosteoses already present results in a single tarsal bone mass excluding the talus and lateral cuneiform (Fig. 4*G*).

In the younger patients, most of the toes appear to have only 2 phalanges (Fig. 4*A* and 5*A*). Case 7 (Fig. 5*A*), at the age of 8 years, illustrates absence of fusion at the distal interphalangeal joints, while a later roentgenogram of the same case, at the age of 26 years 6 months (Fig. 5*B*), shows fusion between these joints. This fusion of the interphalangeal joints is also observed in Case 8 and Case 9 (Fig. 4, *G* and *H*). Only Case 7 shows fusion of the phalanges with the metatarsals. There is fusion of the proximal phalanges of the 3rd and 4th toes with the heads of the corresponding metatarsals on the left (Fig. 6*B*), while on the right (Fig. 5*B*) there is fusion of the phalanges of the 2nd and 3rd toes with the heads of their corresponding metatarsals.

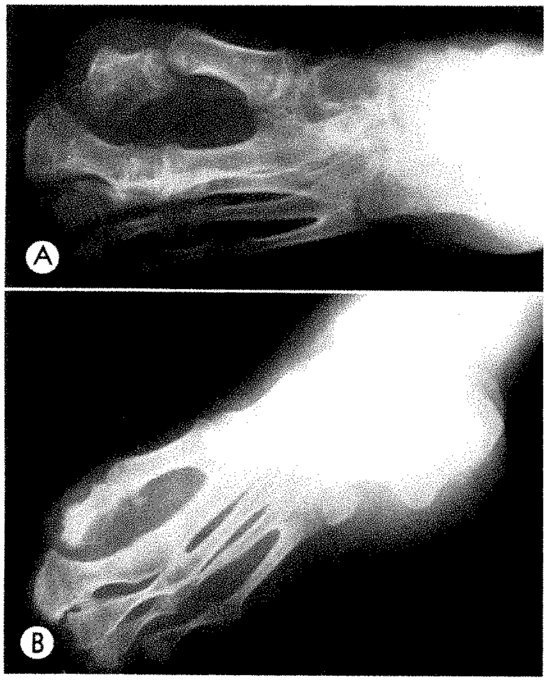

Fig. 5. *Case 7.* Right foot. (*A*) At age 8 years, the terminal phalanges are separate and no evidence of fusion of the metatarso-phalangeal joints is noted. (*B*) At age 26 years 6 months, fusion of the 2nd and 3rd metatarso-phalangeal joints, obliteration of the terminal interphalangeal joints and a "pseudo-articulation" between the phalangeal masses of the 2nd and 3rd toes are demonstrated.

These findings are not present on an earlier roentgenogram at the age of 8 years (Fig. 5*A* and 6*A*). In Case 5 (Fig. 4*C*) there is fusion identified between the terminal phalanges of the 2nd, 3rd, and 4th toes, which is an uncommon finding in Apert's syndrome. The only other case in our series showing fusion between the phalanges is Case 9 (Fig. 4*H*), where it is present between the bases of the fused phalanges of the 3rd and 4th toes.

Fusion between the 1st and 2nd metatarsals, best illustrated by Figure 5*B*, is present in 8 of 9 cases, with Case 6 the exception. This latter case has fusion between the 4th and 5th metatarsals which was not present shortly after birth.

Failure of normal tubulation of the bones is well seen in the roentgenographic illustrations.

In the older patients, there are bony outgrowths from the phalanges which have been previously described.[5]

Bone age is retarded, in particular in the development of the cuneiforms (Fig. 4, *B* and *C*).

"Pseudo-articulations" are present in the older patients between the phalanges and metatarsals of the different toes (Fig. 4*G*, 5*B*, and 6*B*). In Case 7, at the age of 26 years 6 months, "pseudo-articulations" between the 2nd and 3rd phalanges and between the 2nd and 3rd metatarsals have developed since the age of 8 years (Fig. 5, *A* and *B;* and 6, *A* and *B*).

An interesting finding in the 4 oldest cases is the presence of an unusually large os peroneum. The os peroneum, an accessory bone of the foot, occurs in 7 to 11.7 per cent of normal individuals,[6,7,9] and is located in the terminal portion of the tendon of the peroneus longus muscle. It is usually less than 5 mm. in greatest dimension. The largest os peroneum identified by Dwight[6] in his large series, measured approximately 1×2 cm. This center is not present in Case 7 at the age of 8 years, while at the age of 26 years 6 months it is clearly seen near the base of the 5th metatarsal (Fig. 6, *A* and *B*). The presence of a large os peroneum, measuring up to 2 cm., in the 4 oldest patients suggests that it is a common finding in older patients with Apert's syndrome.

The abnormal roentgenographic findings in most cases are bilaterally symmetric.

SUMMARY

The feet in Apert's syndrome show a definite pattern of fusion which occurs in the following order: calcaneus with cuboid, lateral cuneiform with third metatarsal, navicular with medial cuneiform, and phalanges at the interphalangeal joints. Also present in the feet of older patients are "pseudoarticulations," well developed ossa peronei, and bony outgrowths from the phalanges.

A description of progressive synosteoses of the bones of the hands and cervical spine has also been presented. In our review of the literature of Apert's syndrome, no mention of progressive synosteoses has been noted.

Because of the generalized changes and the increasing deformities with age, acrocephalosyndactyly does not seem a particularly good descriptive term for this disease, and in its place progressive synosteosis with syndactyly is suggested. Progressive synosteosis with syndactyly is more inclusive since the acrocephaly is for the most part explained by premature synosteosis.

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THE SILVER SYNDROME: CONGENITAL ASYMMETRY, SHORT STATURE AND VARIATIONS IN SEXUAL DEVELOPMENT*

ROENTGEN FEATURES

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IN 1953, Silver *et al.*[16] reported 2 children with a syndrome characterized by shortness of stature, hemihypertrophy and variations in sexual development. Subsequently, other children with similar manifestations were reported by various authors and, in 1964, Silver[17] summarized the findings in 29 examples of the syndrome. Two additional patients have been described by Reister and Scherz[14] and were not included in Silver's review.

The roentgen findings in this syndrome have received no particular attention in previous reports, nor has the syndrome been discussed in the radiologic literature. The purpose of the authors is to report 2 additional patients with the syndrome and to discuss and illustrate the characteristic roentgen features.

REPORT OF CASES

CASE I. An 11 year old female was admitted to the hospital with an acute gastroenteritis. Little was known of her family history as she was adopted at 3 months of age. Although she had been delivered at term, her birth weight was 1,620 gm. (3 lb., 9 oz.). She was discharged from the premature nursery at 3 months of age when her weight was 5 lb. Growth retardation and slow weight gain persisted and studies elsewhere were said to have been noncontributory. The appearance of breast development and axillary and pubic hair was noted at 8 years of age. Menarche occurred at 9 years of age and irregular menses had recurred infrequently since that time.

On examination the patient presented a markedly linear configuration with a stature which was small in relation to her mature facial appearance. There was complete asymmetry with the left half of the face, trunk and extremities smaller than the right. Her weight was 24 kg. (53 lb.), more than 3 standard deviations below the mean for her age. Her height was 140 cm. The distance from the anterior superior spine to the medial malleolus was: left 68 cm. and right 71 cm. The acromio-finger tip distance was 57 cm. on the left and 60 cm. on the right. The mid-thigh circumference was 26 cm. on the left and 30 cm. on the right. The mid-biceps circumference was 41 cm. on the left and 45 cm. on the right. Muscular development was obviously greater on the right half of the entire body. The facies presented a triangular appearance with the cranial vault disproportionately large for the facial area. The cranial vault was dolichocephalic and the facial area was long and tapered to a small chin. The dental arch was crowded; dental maturation was compatible with the chronologic age. There was a high arched palate. A left internal strabismus was present. The ocular fundi were normal. No abnormality of the heart or lungs was noted. The blood pressure was 100/80. The abdomen was soft; the liver edge was palpable 3 cm. and the spleen 2 cm. below the costal margins. Both kidneys were readily felt but were not considered to be enlarged. There was a moderate growth of axillary and pubic hair. The clitoris was of normal size and the vaginal mucous membrane appeared well estrogenized. The breasts revealed advanced pubescent development. Acne was present over the forehead and nose. A moderate degree of hypertrichosis was present, most marked over the extremities and the dorsal trunk. Bilateral pes cavus was noted. The 5th digits were small and incurved and the middle fingers appeared disproportionately long. A 24 hour urinalysis for gonadotropins was positive at a level of 50 units.

Roentgen examination of the chest showed a definite asymmetry of the thorax (Fig. 1). The right hemithorax was larger than the left. The right ribs were somewhat larger than the left and there was an increased caudal pitch to the left ribs. The right upper and lower extremities were larger and longer than the left (Fig. 2). There was an increase of soft tissue shadow on both right extremities compared to those of the left side. There was no appreciable difference in bone maturation between the two sides. The bone age at the hands and wrists corresponded to a chronologic age of about 14 (Greulich and Pyle). This represented a definite advance in osseous maturation for this patient. Both 5th digits were short and incurved (Fig. 3). The middle phalanges of these fingers were hypoplastic. Mild radial inclination of the distal ends of the ulnas was also noted. The pelvis was markedly asymmetric (Fig. 4). Both innominate bones were small but the right was obviously larger than the left. There was bilateral pes cavus. The skull and spine did not show any appreciable asymmetry.

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Case II. The patient, a 15 year old white male, had been observed off and on over a period of 8 years because of hemihypertrophy. His birth weight had been 3 lb., 6 oz. and a definite asymmetry between the two sides of his body had been noted since he was 8 weeks of age. On this admission he appeared quite short in stature. The left side of the body was larger than the right. The left leg was 2 inches longer than the right and the left arm was ½ inch longer than the right. Both left extremities were larger in girth than those of the right. Both 5th digits showed incurving of the distal phalanx with some bulbous enlargement of the distal soft tissues.

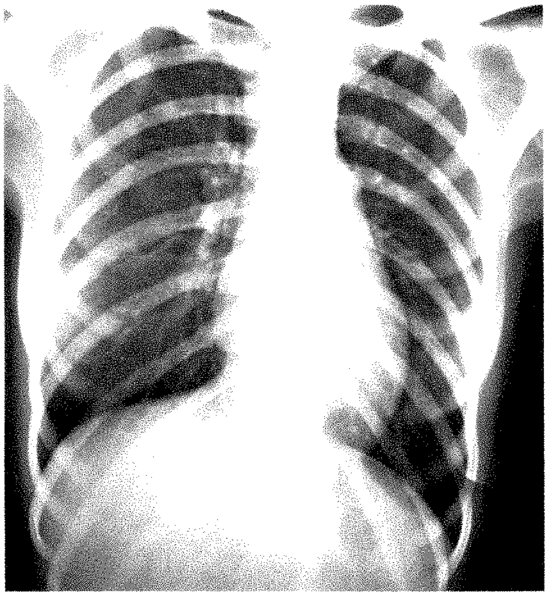

Fig. 1. Case I. There is a significant asymmetry of the thorax. The right hemithorax is larger than the left. The caliber of the right ribs is slightly larger than that of the left ribs and the left ribs show an increased caudal pitch.

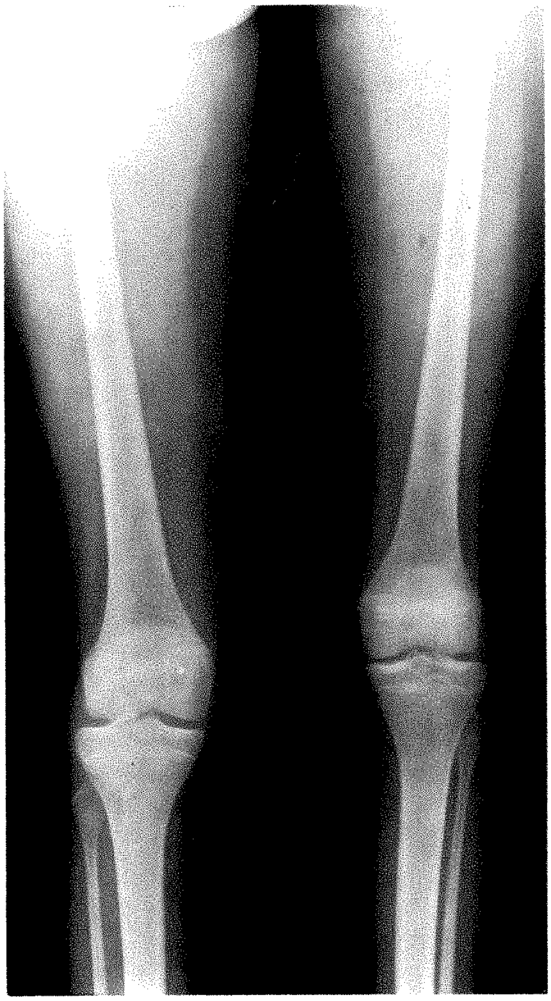

Fig. 2. Case I. The right lower extremity is longer than the left and is slightly larger in caliber. The soft tissues of the right thigh can be seen to be larger than those of the left.

There was an abnormality of the right foot associated with syndactylism. Areas of cafe-au-lait pigmentation were noted on the abdomen and back. The penis and testicles were unusually small. The scrotal folds did not appear to be fully developed. The patient's mentality was considered to be average for his age.

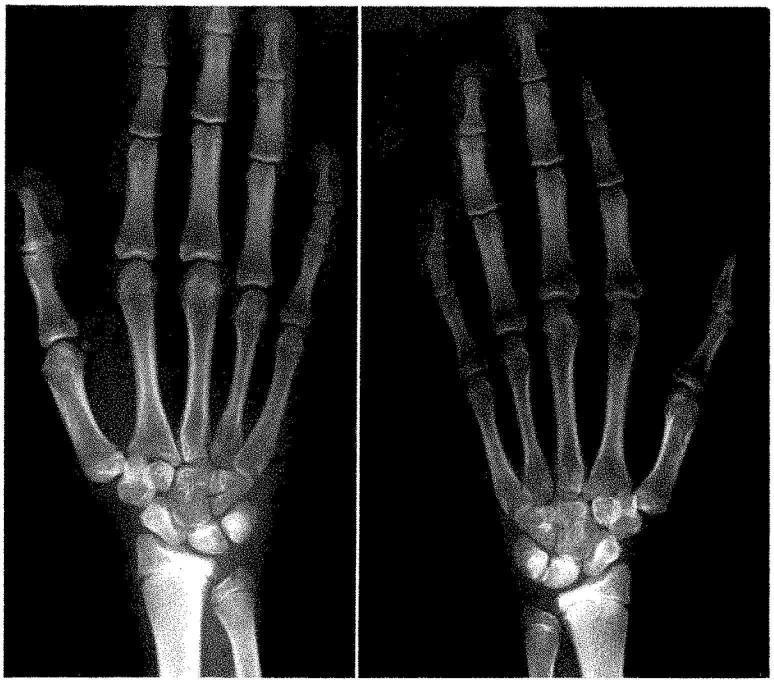

Fig. 3. Case i. The middle phalanges of both 5th fingers are hypoplastic with shortening and incurving of these digits. Osseous maturation is the same on both sides and is advanced for the patient's chronologic age. Some radial inclination of the distal ulna is also noted bilaterally.

Roentgen examination showed a deformity of the right big toe with an abnormality of growth of the proximal phalanx which resulted in medial deviation of the toe (Fig. 5). This appeared to be due to early fusion of the medial portion of the epiphysis. The middle and distal phalanges of the 5th toes appeared to be fused. The lesser toes were held in a flexed position. The left upper and lower extremities were larger and longer than the right. Both 5th digits of the hands were curved. The incurving involved the shaft of the distal phalanx. The epiphyses of these distal phalanges on both sides were thicker than usual and thicker than those of the other distal phalanges. There was no hypoplasia of the middle phalanges. The abnormality of the 5th digits was classic for Kirner's deformity (Fig. 6). The bone age at the hands and wrists was the same on both sides and corresponded to a chronologic age between 14 and 15 (Greulich and Pyle). Roentgen examination of the hands and wrists at earlier ages (7 and 9 years) was reported to have shown some retardation of maturation but this had apparently accelerated to a point where the bone age and chronologic age were about the same at the age of 15.

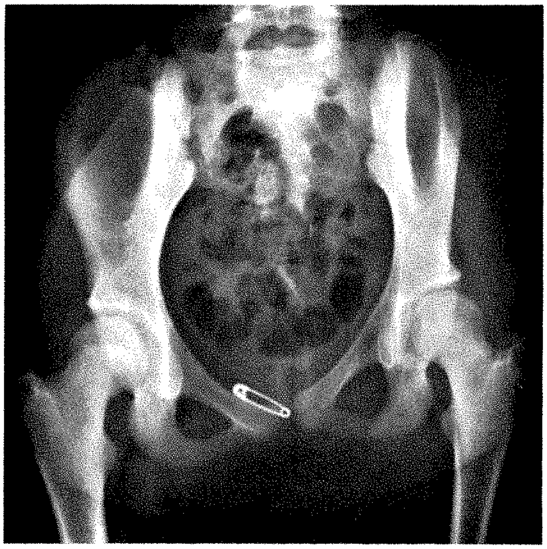

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Fig. 4. Case i. There is a significant asymmetry of the pelvis. The right innominate bone is larger than the left with a resulting pelvic obliquity. A difference in caliber between the upper femora can also be appreciated.

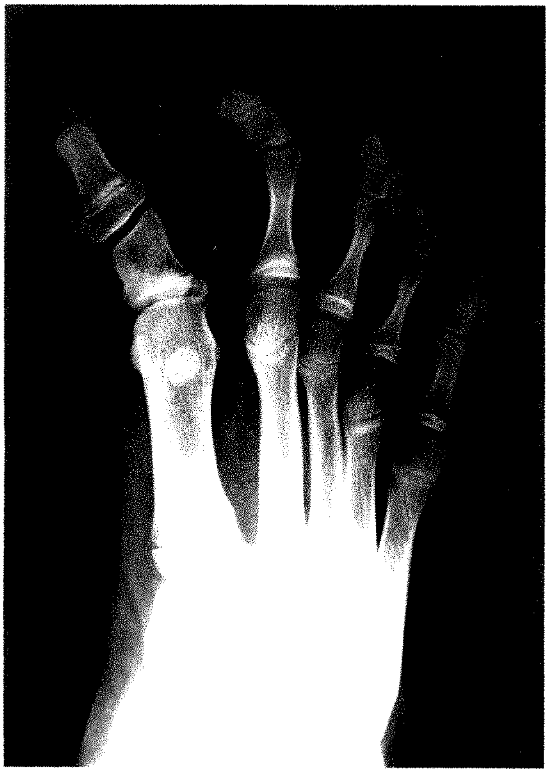

Fig. 5. Case II. The right big toe is deviated medially. This is apparently the result of premature fusion of the medial portion of the epiphysis. The 2nd, 3rd, and 4th toes are held in flexion and the middle and distal phalanges of the 5th toe are fused.

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DISCUSSION

The patients described by Silver appear to belong to a larger category of "low birth weight dwarfism" or "intrauterine growth retardation" of undetermined origin. Numerous congenital stigmata and anomalies are found to be associated with this type of primordial small stature and such individuals have been variously classified by several authors, dependent upon certain dominant features which appear to set each group apart. Until more is known about the etiologic factors responsible for the growth retardation, however, the eponymic subcategories are largely arbitrary. Thus, Russell[15] has described a syndrome of "in-

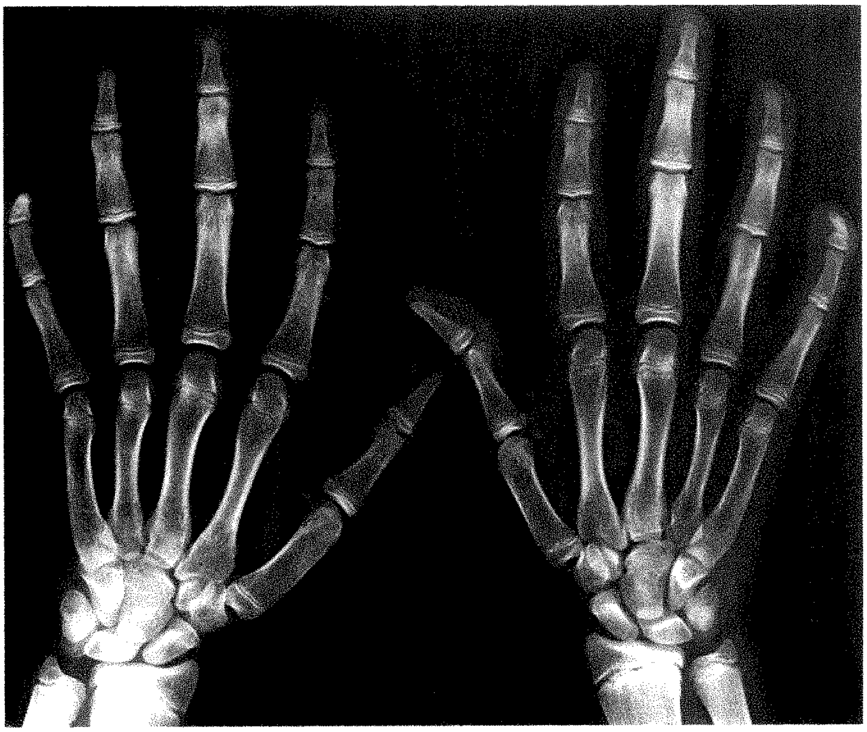

Fig. 6. Case II. The shafts of the distal phalanges of the 5th fingers are curved radially and ventrally. The epiphyses of these phalanges are thicker than normal and are thicker than the epiphyses of the other distal phalanges. Associated with this osseous deformity is a bulbous swelling of the soft tissues about the deformed phalanges. The appearance is classic for Kirner's deformity. The bone age is the same on both sides.

trauterine dwarfism recognizable at birth with cranio-facial dyostosis, disproportionately short arms and other anomalies." Of 5 children in his report, 2 presented congenital asymmetry and have been included in Silver's review as instances of the Silver syndrome. Bloom[4] has described a low birth weight type dwarf with congenital telangiectatic erythema resembling lupus erythematosus and Cockayne[5] has reported a variety of dwarf with retinal atrophy and deafness. The terms "progeroid dwarf" and the "bird headed dwarf" have been employed to describe congenital short stature with a facies suggestive of premature senility with a prominent brow and nose and turned down corners of the mouth.

Silver lists the manifestations of his syndrome as: significant asymmetry, shortness of stature, small size despite being born at term, cafe-au-lait areas of pigmentation of the skin, unusually short 5th fingers, triangular shape to the face, turned down corners of the mouth and syndactylism of the toes. Variation in the pattern of sexual development may include: elevated urinary gonadotropin or premature estrogenization of the vaginal or urethral mucosa or retarded skeletal age in relation to sexual development. It is to be noted that no one of the patients described in his review presented all of the above manifestations. The stigmata of the syndrome may be divided into major and minor manifestations however, and Silver[18] is of the opinion that any child with three of the major manifestations definitely has the syndrome, particularly if there are one or more of the minor manifestations present (Table 1). Significant asymmetry was present in 78 per cent of the patients and was variable in extent and degree. Shortness of stature was present in 93 per cent and usually was associated with low weight and small size at birth. Variation in the pattern of sexual development was present in only 34 per cent of the children. Urinary gonadotropins were elevated in 5 children prior to 9 years of age. Four of the children, all girls, exhibited clinical evidence of precocious sexual development. The so-called "shark mouth" with elevated philtrum and turned down corners was present in 62 per cent of the series. There was no sex or ethnic preponderance. No familial cases were found.

TABLE I

SILVER'S CRITERIA FOR THE SYNDROME

Major Manifestations

1. Shortness of stature
2. Significant asymmetry
3. Small size despite being born at term
4. Variations in the pattern of sexual development
    a) Elevated urinary gonadotropins
    b) Early sexual development
    c) Premature estrogenization of urethral or vaginal mucosa
    d) Retarded bone age in relation to sexual development

Minor Manifestations

1. Short 5th fingers
2. Incurved 5th fingers
3. Triangular shape to face
4. Turned-down corners of the mouth
5. Cafe-au-lait areas of pigmentation of skin
6. Syndactylism

After Reister and Scherz.[14]

ROENTGEN FEATURES

The outstanding roentgen feature of this syndrome is hemihypertrophy. In most cases the so-called hypertrophy involves practically one entire side of the patient. In some cases, however, the hemihypertrophy may be segmental or partial, involving only the skull, the spine or an extremity. Review of the previously reported cases indicates that in some, at least for a while, epiphyseal development is retarded on both sides. In others bilateral epiphyseal development has been normal for the patient's age. Rarely, epiphyseal development has been said to be retarded on the small side and normal on the large side and in at least 2 cases osseous maturation has been reported as advanced. In several instances, however, no comment regarding bone age is offered and, in most cases where bone age is stated to be retarded, no information is presented to indicate how such an estimate was achieved. On the basis of all available material pertinent to this subject, there does not appear to be any consistent pattern of bone maturation, but more detailed roentgenographic study of patients with this syndrome in the future may alter this impression. It is worth noting that in Case I of this report there was no inconsistency between the pattern of sexual maturation and epiphyseal development. Both were advanced. It is also noteworthy that early retardation of bone maturation in Case II was eventually overcome by the age of 15 years.

The hemihypertrophy is generally observed early in the patient's life, usually within the first few months. Soft tissue enlargement is present in addition to the increase in bone size. The etiology of hemihypertrophy is unknown, but it is generally considered to represent a developmental anomaly. The fact that the condition is often found in association with other congenital anomalies adds credence to this view. Two patients with Silver's syndrome reported by Reister and Scherz[14] have had chromosomal studies which revealed 46 normal appearing chromosomes. These studies, however, were limited to the peripheral blood leukocytes. On the other hand, Ferrier *et al.*[7] have reported a case of congenital hemihypertrophy with features consistent with Silver's syndrome in which there was a diploid-triploid mosaicism. In this case there were diploid and triploid cells in the skin and fascia lata, while the bone marrow and peripheral leukocytes were diploid only. Ellis *et al.*[6] have also reported a patient with congenital asymmetry associated with diploid-triploid mosaicism. In their case too, triploid and diploid cells were found in the skin, but only diploid cells were recovered from the blood. It is significant that in both of the above mentioned cases diploid and triploid cells were found in equal proportions in skin from both sides of the body. Hook and Yunis[9] have recently described a patient with congenital asymmetry associated with trisomy 18 mosaicism. Cells with a normal chromosome complement and cells with a trisomy 18 pattern were recovered from skin tissue, bone marrow and peripheral blood from both sides of the body. These authors have suggested that the finding of mosaicism on both sides of the body in patients with congenital asymmetry may be due to embryonic migration of these mesenchymal cells across the midline after formation of an asymmetric fetus. At any rate, the possibility of chromosomal mosaicism should be more fully investigated in patients with congenital asymmetry with or without features of Silver's syndrome.

Approximately 120 cases of congenital hemihypertrophy have been reported, most of them unrelated to intrauterine growth retardation. A significant association between congenital hemihypertrophy and malignancy has been uncovered. Twelve cases demonstrating this association have been reported. Of these, 10 have had Wilms' tumors and of these 7 were on the hypertrophied side while 3 were on the contralateral side. The patients were between 1 and 4 years of age when these tumors were discovered. In all cases the patients had been examined because of their asymmetry before the tumors were noted. Benson *et al.*[2] have reported that among a total of 6 children seen by them with hemihypertrophy not due to vascular malformation or neurofibromatosis, Wilms' tumors developed in 4. Hutchinson[10] has described a patient with congenital hemihypertrophy with adrenal hyperplasia and Harwood and O'Flynn[8] have reported sexual precocity due to adrenal adenocarcinoma in a 6 year old boy with hemihypertrophy. Lewis and Geschickter[13] have reported a right sided suprarenal neuroblastoma in a 4 year old girl with ipsilateral hemihypertrophy. None of the reported cases of Silver's syndrome has been associated with malignancy.

Short incurved 5th fingers are a relatively consistent feature of the syndrome. Where there has been some comment regarding the bones of these digits, a hypoplasia of the middle phalanges has been reported, such as illustrated in Case I of this report. In many cases, however, the description of the fingers has been purely clinical without any evaluation of the bone structure. It was surprising, therefore, to find in Case II a typical Kirner deformity. This deformity, first described by Kirner[12] in 1927, also produces an incurved 5th finger. Recent reports on this deformity have been made by Kaufmann and Taillard,[11] Taybi,[19] and Blank and Girdany.[3] This abnormality is characterized by bilateral incurving of the distal phalanges of the 5th fingers. The osseous deformity is preceded by a globular swelling of the soft tissues of the distal phalanx. The shaft of the terminal phalanx deviates radially and in the palmar direction. The epiphysis remains in the normal position but it is usually much thicker than normal. During development, the metaphysis of this phalanx is poorly defined and the epiphyseal line is widened. Fusion of the shaft with the epiphysis usually occurs at the normal time, but the shaft maintains its deviated position and the bulbous enlargement of the adjacent soft tissue persists. The deformity may be familial. Blank and Girdany[3] have described 7 cases in 3 generations of one family.

Other less frequent and less characteristic skeletal abnormalities have been mentioned as occurring in some cases of the syndrome.[1] These include poorly formed thoracic vertebrae which differ from those seen in the ordinary type of hemivertebra, surface irregularities of the lumbar vertebrae, hypoplasia of various phalanges and hypoplasia of the sacrum and coccyx.

SUMMARY

1. Two cases of the Silver syndrome are reported. The major characteristics of this syndrome are shortness of stature, significant asymmetry, small size at birth despite being born at term and variations in the pattern of sexual development. Minor manifestations include short incurved 5th fingers, triangular shape to the face, turned-down corners of the mouth, cafe-au-lait areas of pigmentation of the skin and syndactylism.

2. The most consistent roentgen features are congenital hemihypertrophy and hypoplasia of the middle phalanx of the 5th fingers. One case is reported in which there was a Kirner's deformity of the 5th fingers. Other less frequent skeletal abnormalities which have been noted in some cases are mentioned.

3. The syndrome may be considered one category of low birth weight dwarfism with special congenital stigmata which set it apart from other varieties of intrauterine dwarfism.

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THE ROENTGENOGRAPHIC MANIFESTATIONS OF THE RUBELLA SYNDROME IN NEWBORN INFANTS*

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IN THE spring of 1964 a severe rubella epidemic occurred in Houston, Texas, and in a number of other large communities in the United States. Infants born of mothers who had rubella in the first trimester of pregnancy during this virulent epidemic had several unusual findings, consisting of growth retardation, hepatosplenomegaly, thrombocytopenic purpura, large anterior fontanel and peculiar osseous lesions, in addition to congenital heart disease, eye defects, and deafness. Although deafness, congenital cataracts and cardiac anomalies have been recognized as prenatal manifestations of rubella[2,4] for a number of years, the clinical syndrome seen in this group of patients is unique.

Of special interest to the radiologist are the osseous abnormalities which accompany this rubella infection. In September, 1964, Dr. Reilly[12] reported 3 infants with unexplained osseous lesions to the Society for Pediatric Radiology. Although the association of these lesions with maternal rubella was not appreciated; in retrospect, this was the first scientific report of bone changes with rubella. The first published account of lesions of rubella infection demonstrating osseous defects was that reported by Rudolph *et al.*[14] and this in turn was followed by sporadic reports of similar bone lesions.[6,9,10,11] In one of these reports, unexplained fractures of the humerus were mentioned[6] and in another attention was called to the poor mineralization of the calvarium.[9] Because of the virtually pathognomonic appearance of the bone lesions in infants with the rubella syndrome, a more detailed report covering a large number of cases is of value in more firmly establishing the roentgenographic features of not only the osseous abnormalities but other radiologically detectable lesions which accompany this condition.

A complete discussion of the clinical findings of 81 infants reviewed in this study has been previously published.[16]

CLINICAL FINDINGS

Clinically, these infants are divided into 3 groups (Table I). Group II comprises infants with the usual expected rubella sequelae, including congenital heart disease and congenital cataracts. Group I consists of the "expanded rubella syndrome" found in most of the infants on whom the present interest is centered. This group includes those infants with a positive maternal history of rubella and the usual rubella defects of cataracts and heart anomalies, as well as the additional features of growth retardation, thrombocytopenic purpura,

Table I

CLINICAL CLASSIFICATION OF CONGENITAL RUBELLA SYNDROME

Group I	Positive history of maternal rubella manifestations of the "expanded" rubella syndrome (intrauterine growth retardation, purpura and thrombocytopenia, hepatosplenomegaly, bone changes, full fontanel, cardiac defects and eye defects)
Group II	Congenital cardiac and eye defects associated with a positive or negative history of maternal rubella
Group III	Positive history of maternal rubella with a presumably normal baby

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Table II

CLINICAL FINDINGS IN 81 INFANTS WITH CONGENITAL RUBELLA SYNDROME

	Group I "Expanded" Rubella Syndrome	Group II Classical Rubella Syndrome	Group III History of Maternal Rubella—Presumably Normal Baby
No. of Infants	34	37	10
Males	26	23	7
Females	8	14	3
Mean Gestational Age (wk.)	40.1	39.8	39.8
Mean Birth Weight (gm.)	2,178	2,533	3,327
Purpura	78%	0	0
Thrombocytopenia (<140,000)	100%	0	0
Hepatomegaly	85%	81%	20%
Splenomegaly	76%	62%	10%
Cardiac Defects	78%	86%	0
Eye Defects	41%	54%	0
Full Fontanel	69%	43%	0
Mortality Rate	32%	8%	0

hepatosplenomegaly, a full fontanel and bone defects detected by roentgenographic examination. Group III includes normal infants who have a positive maternal history of rubella.

The clinical findings in 81 infants with the congenital rubella syndrome are listed in Table II. In Group I the male patients outnumbered the female by a ratio of more than 3–1, and although the mean birth rate of the Group I infants suggested prematurity, the mean gestational age was essentially normal. This combination of normal gestational time and a low birth weight indicates intrauterine growth retardation or fetal malnutrition.

The purpuric lesions evident at delivery were seen in all of the Group I infants and in none of the other groups. The lesions were distributed generally over the body, but were especially pronounced on the face. Thrombocytopenia was responsible for the purpura, the platelet counts being less than 140,000 in all cases. The purpura and thrombocytopenia cleared rapidly within 1 to 2 weeks and did not recur.

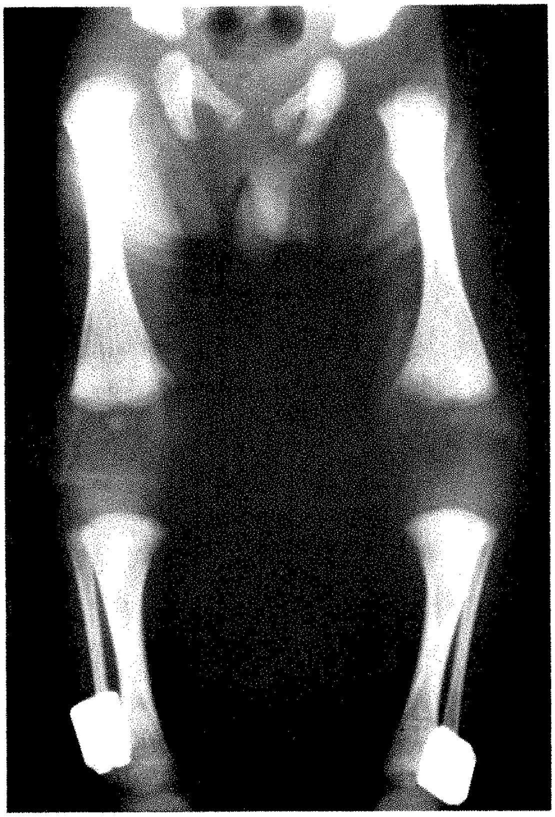
Central nervous system involvement was manifested in many of the infants of both Groups I and II by the presence of a full anterior fontanel, pleocytosis, increased protein content of the cerebral spinal fluid and abnormal electroencephalograms.

The mortality rate in the Group I infants has been especially high, the infants developing poorly both nutritionally and neurologically.

Positive viral cultures have been obtained from the throat, stool, urine, blood, cerebral spinal fluid and bone marrow specimens.[14] Viral isolation has been highest in the Group I infants, and less frequent in Group II. Fifty per cent of the Group III infants have had positive viral cultures. Since most of these infants continue to excrete virus after birth, there is the constant danger of infection developing in other infants in the nursery, as well as in the personnel handling of the infant. In addition, the likelihood of further damage to the tissues of these infants occurring from their continual infection is to be considered.

ROENTGENOGRAPHIC FEATURES

The positive roentgenographic features of the expanded rubella syndrome are found in the skull, heart and the long bones of upper and lower extremities.[17] The roentgenographic features in the long bones are especially interesting and in our opinion, pathognomonic, especially when specific



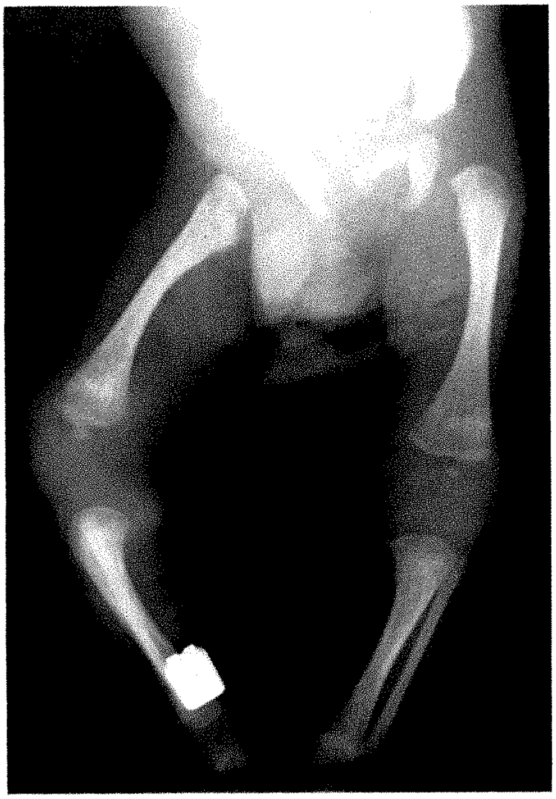


Fig. 1. One day old male infant with maternal history of rubella. Longitudinal areas of radiolucency and increased density occupy the metaphyses of the lower extremity. The provisional zones of calcification are poorly mineralized and are irregular.

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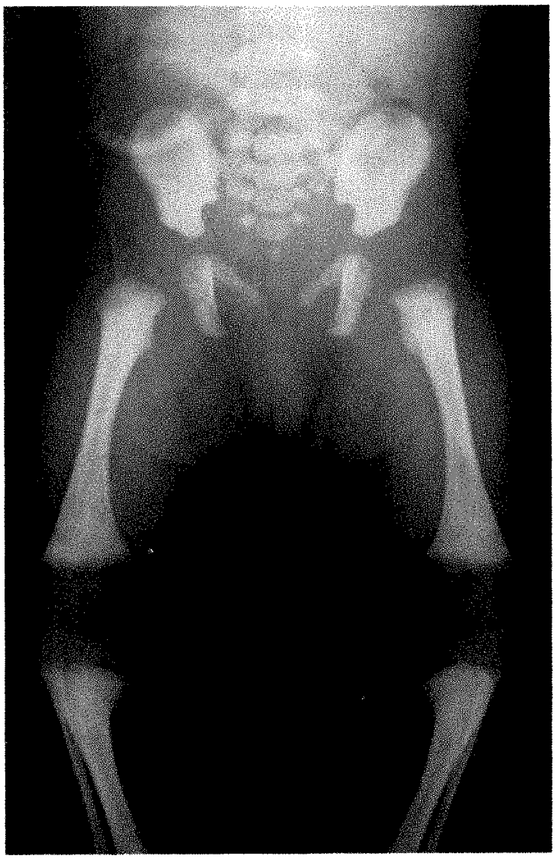

Fig. 3. Two day old female with maternal history of rubella. The growth plates are extremely irregular and poorly mineralized. Longitudinal areas of radiolucency occupy the metaphyses.

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Fig. 2. One day old male infant with maternal history of rubella. Alteration of trabecular pattern of the metaphyses characteristic of the rubella syndrome is present.

changes are present. In the newborn these changes consist of a poorly defined provisional zone of calcification and coarsening of the metaphyseal trabeculae with longitudinal areas of radiolucency and sclerosis. All of the metaphyseal areas are involved but are especially prominent at the proximal tibial and distal femoral metaphyses (Fig. 1, 2, 3, 4 and 5). The shafts of the bones are essentially normal. If the infant survives and grows satisfactorily, the metaphyseal trabecular pattern returns to normal and concomitant with this the provisional zones of calcification show an increase in density and smoothness. The rapidity and completeness of the regressive bone changes appear to correlate closely with the clinical well-being of the infant. In those infants who thrive and maintain normal growth, the bones appear normal in 1 to 3 months except for residual growth disturbance lines (Fig. 6, *A* and *B*). Follow-up roentgenographic studies of many rubella babies who have not shown normal development have disclosed variations in the roentgenographic pattern. In many of the infants, there is persistence of the irregular growth plate and altered trabeculation of the metaphyses but the provisional zones of calcification become dense and may even be unusually thick and sclerotic (Fig. 7 and

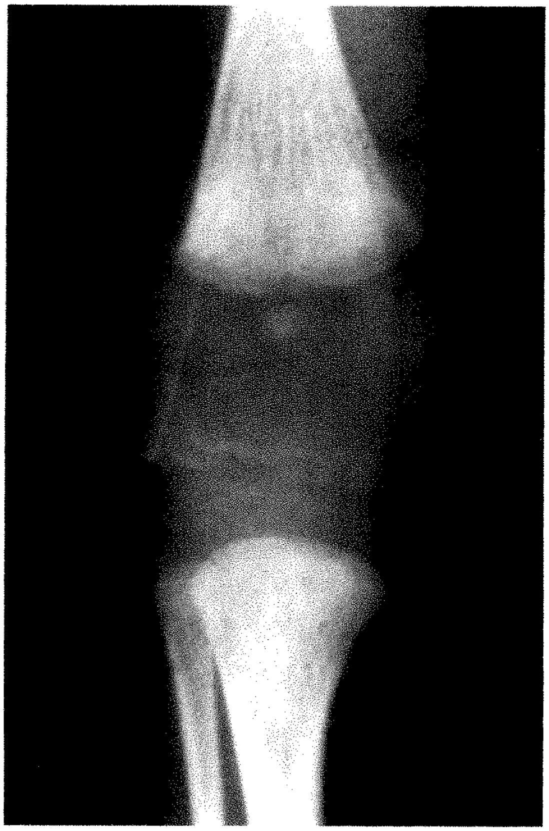

Fig. 4. One day old female with maternal history of rubella. Enlargement of knee roentgenogram shows to better advantage the characteristic longitudinal areas of radiolucency and increased bone density.

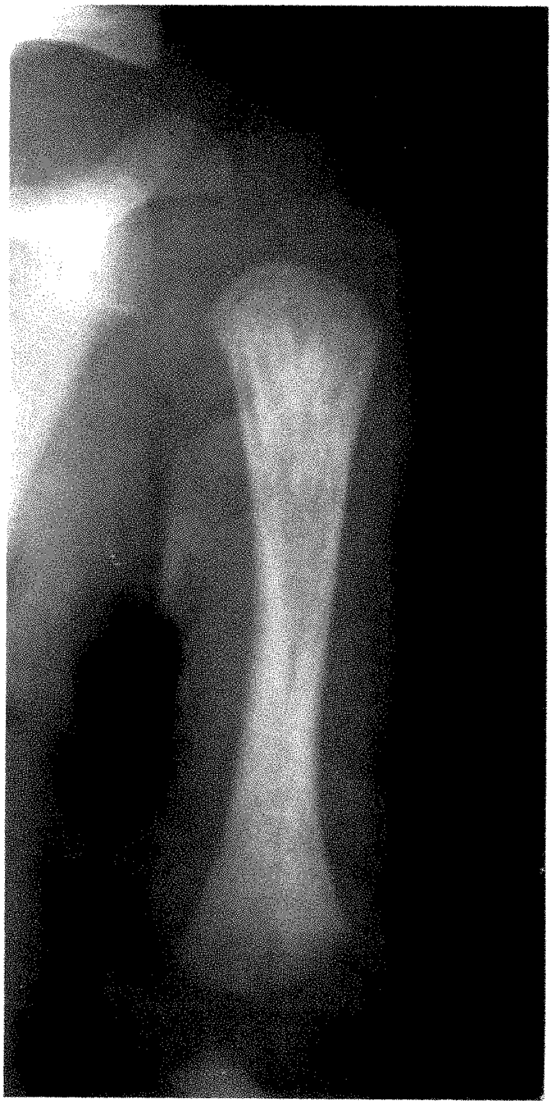

Fig. 5. Upper extremity of "rubella baby" showing alteration of the trabecular pattern of the proximal humeral metaphysis.

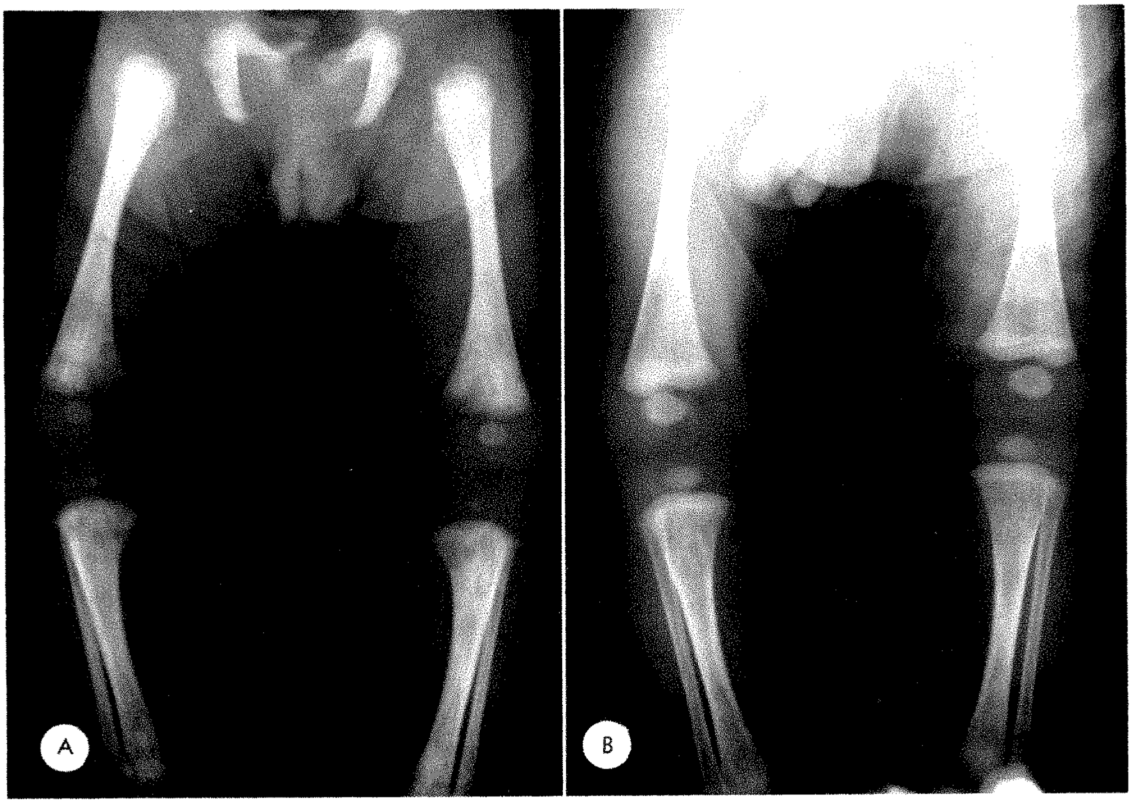

Fig. 6. Osseous changes with growth in infant who did well clinically. (*A*) September 27, 1964. At 2 weeks of age characteristic rubella changes in the metaphyses of the knees are present. (*B*) November 10, 1964. Two months later there has been nearly complete restoration of normal bone.

8). Nonspecific changes may also develop, but, as a rule, are present after the neonatal period. These consist of metaphyseal bands of radiolucency parallel to the growth plates similar to those seen in infants with erythroblastosis fetalis, congenital syphilis, and in infants who fail to thrive because of a number of other conditions (Fig. 9). Small metaphyseal beak-like projections may develop during healing and are most pronounced at the medial aspect of the distal femoral metaphysis (Fig. 10).

All of these changes occurred with greater frequency in the Group I infants and were absent in Group III.

Roentgenograms of the skull showed a remarkably large anterior fontanel in a majority of infants in Group I, and to a lesser extent in Group II infants. The anterior portion of the anterior fontanel in these infants extended well into the frontal bone and involved the metopic suture (Fig. 11,

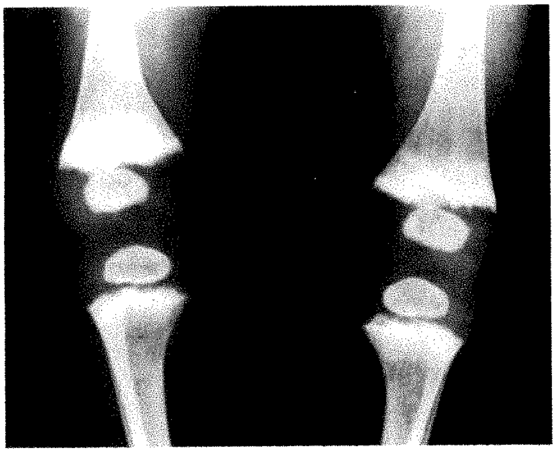

Fig. 7. Late osseous changes. Six month old female infant with maternal history of rubella. Sclerotic and irregular changes affecting the metaphyses of the knees have developed in an infant whose clinical development has been poor. (Courtesy of Dr. Guido Currarino.)

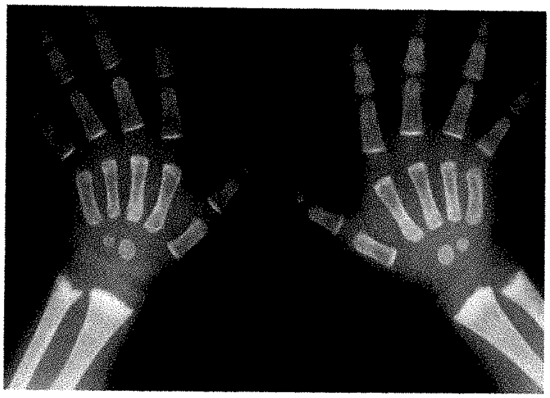

Fig. 8. Ten month old infant with maternal history of rubella. Sclerotic irregular changes affecting the radial and ulnar growth plates are demonstrated.

A and *B*). Suture separation was not a constant feature, but irregular mineralization of the cranial vault in the region of the sutures was frequently seen.

Congenital heart disease commonly occurs in infants whose mothers have had rubella during the first trimester of their pregnancy. The most common anomaly associated with rubella is patent ductus arteriosus[7] (Fig. 12, *A* and *B*). This was also true in the infants included in this review, but a second common lesion was that of pulmonary artery branch stenosis[5,13,18] (Fig. 13, *A* and *B*). Twenty-seven of the rubella patients in this review had patent ductus arteriosus and 26 had pulmonary valvular and/or pulmonary artery branch stenoses. A more detailed account of the cardiac lesions in these infants is to be published.[8]

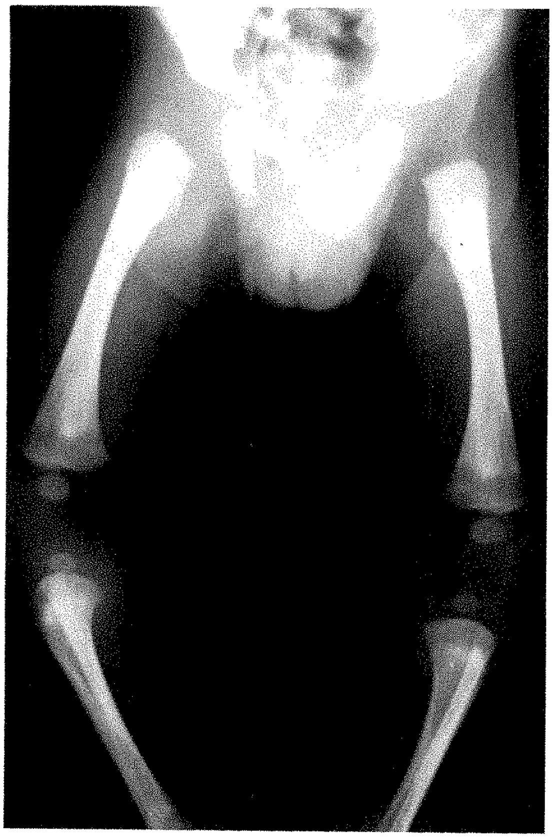

Fig. 9. Nonspecific radiolucent bands are seen in the distal femoral and proximal tibial metaphyses with some residual alteration of the trabecular pattern.

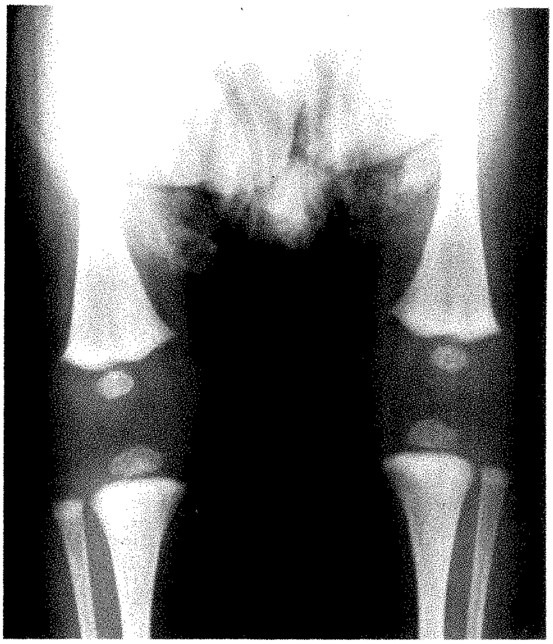

Fig. 10. Two month old female infant with maternal history of rubella. Trabecular pattern shows mild persistent changes and there is nonspecific beaking of the distal femoral metaphyses.

PATHOLOGIC FINDINGS

An understanding of the normal process of endochondral bone formation is necessary to appreciate the nature of the changes which occur in the rubella fetus. At the line of ossification, the cartilage is arbitrarily divided into several zones, the first being the zone of resting cartilage cells. Extending toward the line of ossification, a second zone of proliferating cells is encountered and in this zone irregular clusters of cartilage cells occupy the lacunae. Toward the

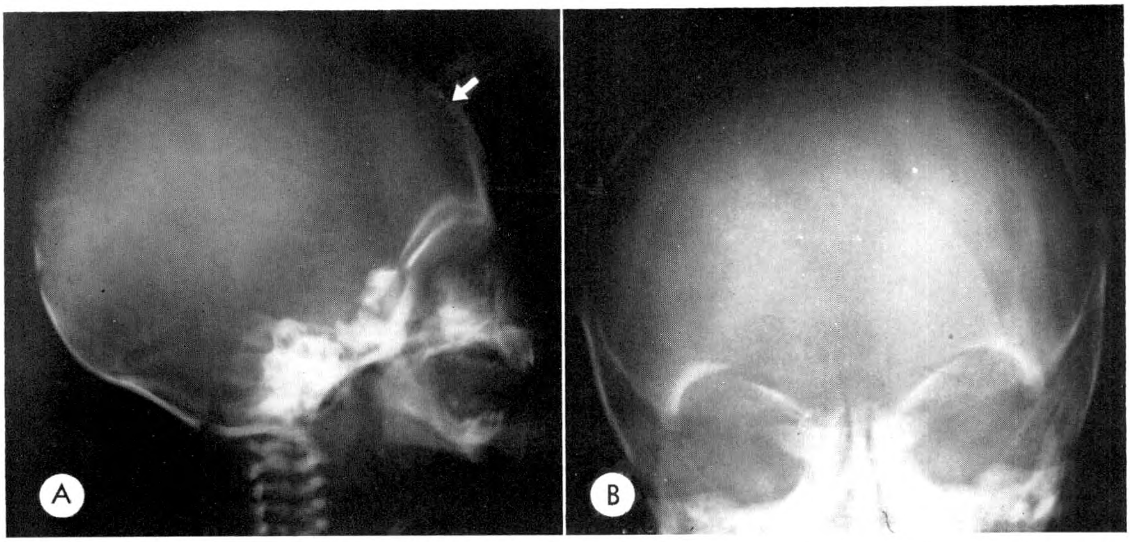

FIG. 11. Skull changes in the rubella syndrome. (*A*) Lateral roentgenogram shows anterior extension of the large anterior fontanel (arrow), as well as prominent sutures and poor mineralization of the calvarium. (*B*) Frontal roentgenogram shows the forward anterior extension of the anterior fontanel to better advantage.

line of ossification, the proliferating cells progress into the zone of mature cartilage cells which lie in orderly columns. Individual lacunae are separated by thin strands of matrix and the columns by a slightly thicker matrix. Bone formation begins with calcification of the hyaline matrix between the cartilage cells which is followed by death of these cells. This in turn is followed by capillary penetration from the marrow space into the column of mature cells, leaving a thin layer of acellular cartilage between the adjacent columns. With the capillary invasion, osteoblasts proliferate and deposit osteoid around the cartilage columns to form spicules which ultimately form the trabeculae of cancellous bone.

Considerable variation in the intensity of bone formation occurs in the infant with transplacental rubella infection. The rib and distal femur show pronounced involvement while the proximal femur shows little change.

Histologically, there are several obvious

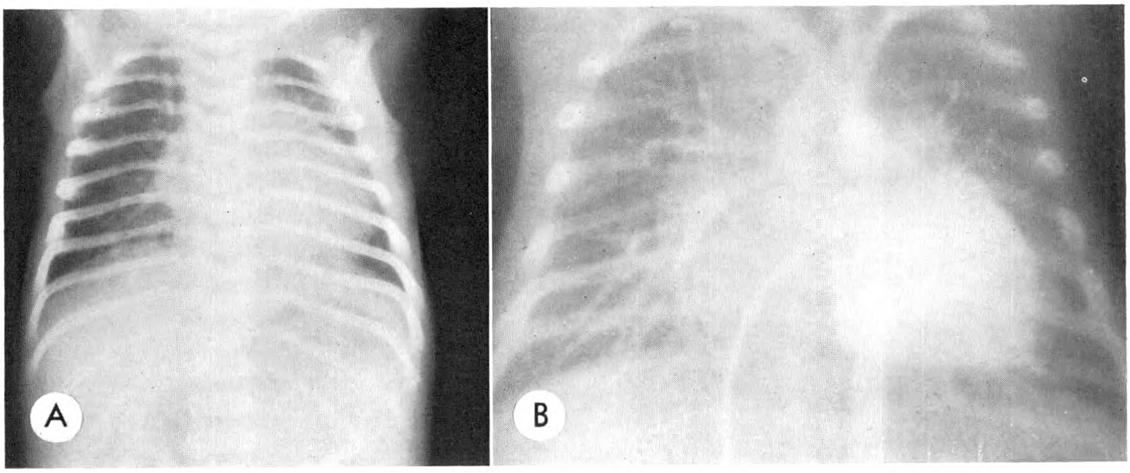

FIG. 12. One day old male infant with maternal history of rubella. (*A*) The heart is slightly enlarged and the pulmonary arterial vessels are increased. (*B*) Angiocardiogram shows injection of contrast medium into the left ventricle with opacification of the aortic arch and pulmonary artery via patent ductus arteriosus.

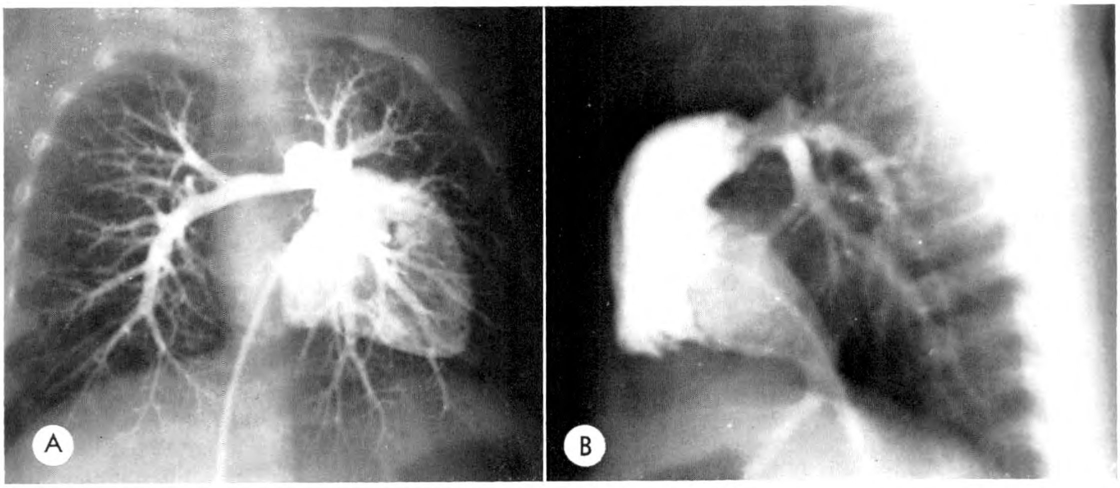

FIG. 13. Pulmonary branch stenosis. Six and one-half month old female with maternal history of rubella. (*A*) Angiocardiogram shows mild stenosis of the right pulmonary artery. (*B*) Lateral study shows evidence of supravalvular pulmonary artery stenosis.

variations which occur in the bone formation of the rubella fetus (Fig. 14). These are: (1) irregular capillary penetration of the zone of mature cartilage; (2) intense osteoblastic activity; (3) poor deposition of osteoid; (4) distorted orientation of the bone spicules; and (5) inadequate calcification of the osteoid spicules. Although there is intense osteoblastic activity, deposits of osteoid are decreased and the hyaline basophilic cartilage persists as small irregular islands into the metaphysis. Progressing further into the metaphysis, much more osteoid is deposited and the cartilage is less prominent. Adjacent to the line of ossification, multiple, poorly calcified, fragments of acellular cartilage and osteoid are present which correspond to the longitudinal areas of radiolucency seen in the metaphyses on roentgenograms.

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DISCUSSION

It has been generally assumed that maternal rubella resulted in fetal damage at critical periods of organogenesis during the first trimester of pregnancy. Whether the clinical and roentgenographic findings pre-

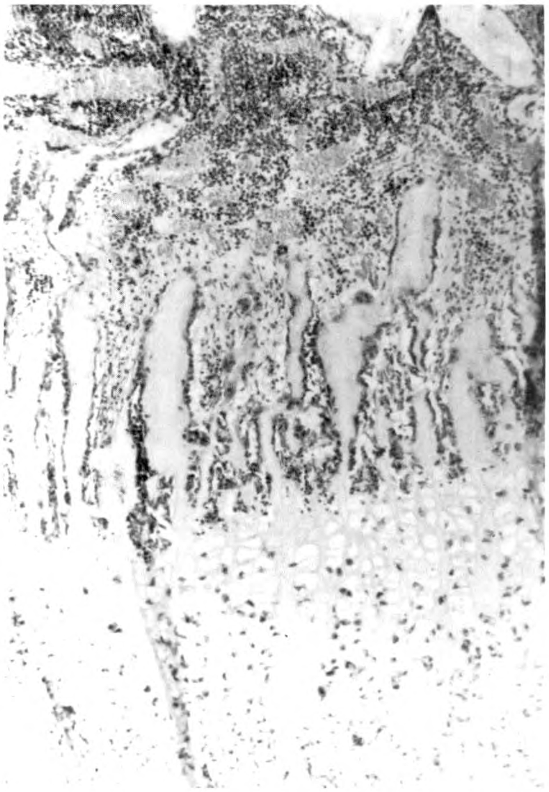

FIG. 14. At the line of ossification, there is only slight variation in depth of the mature cartilage cell layer. The islands of cartilage are large and are surrounded by a dense osteoblastic layer. In the narrow space (at the top of the photomicrograph), the orientation of osteoid islands is distorted. One spicule lies parallel to the line of ossification rather than in the normal perpendicular orientation (hematoxylin-eosin, 64×).

sented in this report are the result of some alteration in the structure or virulence of the rubella virus which allows it to induce changes other than the previously recognized congenital defects is unknown. However, because of the contagious nature of the infant, as well as the isolation of the virus from their stools, urine, cerebral spinal fluid and bone marrow, there is room for speculation regarding a different host-viral relationship than existed on other rubella infections. It is not known how long these infants will excrete the virus or what developmental changes may occur during the postnatal period.

The roentgenographic features affecting the long bones seen in the newborn are of special interest to the radiologist because of their pathognomonic appearance. The characteristic roentgenographic appearance provides diagnostic criteria even in the absence of a positive history of maternal rubella. Although the changes at the growth plate may suggest congenital syphilis, there is no evidence of periosteal reaction. Alkaline hypophosphatasia may be included in the differential diagnosis but the roentgenographic changes are not similar. The localization of the abnormality to the metaphyses with normal diaphyses suggests that the effect on the skeleton occurs later in pregnancy than the first trimester. On the other hand, because healing of the lesions usually occurs quickly in those infants who have follow-up studies, it is conceivable that earlier in fetal life the shafts of the bones, which then represented the metaphyseal areas, were affected but had regained normal structure as intrauterine growth proceeded. Histologic specimens of the metaphyseal regions do not show evidence of osteitis and, consequently, it is impossible to assume that the osseous changes are the direct result of the rubella virus. The changes may well be the result of placental insufficiency or reflect some nutritional disturbance on the growing bone which is more severe or effective in the later weeks of intrauterine development. Roentgenographic changes in the long bones in cytomegalic inclusion disease,[16] as well as in other viral infections,[3] have been reported but not confirmed.

Black-Schaffer[1] described long bone changes in a premature infant whose mother had severe cyanotic cardiovascular abnormalities. The histologic picture of this infant's long bones was similar to the rubella baby, and he suggested that the bone lesions resembled the changes ascribed to experimental inanition and that intrauterine hypoxia may have produced the changes.

In all probability, the roentgenographic findings of the long bones of all the babies having the rubella syndrome are identical in the neonate and in those infants who thrive, and there is regression of the lesions with restoration of normal bone after 1 or 2 months. However, in those infants who do not grow and develop satisfactorily, the altered trabecular pattern persists and is commonly accompanied by a thick sclerotic zone of provisional calcification, deposited irregularly along the irregular growth plate. Another change which is nonspecific is that of radiolucent bands paralleling the growth plate. These bands are seen in erythroblastosis fetalis and in congenital syphilis, and probably reflect an alteration in normal cellular activity. This appearance has not been seen in the rubella baby during the first few days of life. Another nonspecific change is that of beak formations at the metaphyseal edges. This is not specific for the rubella babies but represents alteration in the epiphyseal line at the periosteal attachment.

The large anterior fontanel seen in the rubella baby is also probably a reflection of disturbed bone growth on a nutritional basis and is not thought to represent an inflammatory lesion of the calvarium.

Patent ductus arteriosus and peripheral pulmonary artery stenosis have been the most common cardiac lesions in this group of rubella babies. The cardiac defects have been repaired in a number of these babies and their postoperative recovery has been uneventful.

SUMMARY

During the past year, a large number of newborn infants whose mothers had rubella during the first trimester of pregnancy has been observed and unique clinical and roentgenographic features have been noted. The clinical features consist of growth retardation, thrombocytopenia purpura, hepatosplenomegaly, a large anterior fontanel, congenital heart disease, eye defects and deafness. The unique roentgenographic features, which we feel are pathognomonic of the rubella syndrome, consist of irregularity and poor mineralization of the growth plate, as well as an altered trabecular pattern of the metaphyses of the long bones, particularly at the distal femoral and proximal tibial areas. These osseous abnormalities rapidly disappear in those infants who grow normally but show persistence and even alteration in those who do not thrive.

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RUBELLA EMBRYOPATHY*

ROENTGENOLOGIC FEATURES

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GREGG,[13] in 1941, first reported the relationship between maternal rubella in early pregnancy and congenital cataracts. Subsequent authors, including Swan *et al.*[25] described additional congenital defects attributable to rubella, notably cardiac malformations, microcephaly and deaf mutism. Wesselhoeft,[32] in 1947, reported a statistical analysis of 521 cases from the literature and concluded that the commonest sequelae of maternal rubella were deafness, eye defects and cardiac malformations and that the frequency of these anomalies was approximately equal. Microcephaly, mental retardation and dental defects occurred less commonly.

These retrospective studies suggested that the incidence of congenital defects following maternal rubella was extremely high. In fact, Wesselhoeft's analysis of a series of 49 cases of maternal rubella reported by Swan showed that the incidence of congenital defects was 100 per cent if rubella was contracted during the first 2 months of pregnancy. In contrast, recent prospective studies by Manson *et al.*[19] in 1960, Pitt[20] in 1961, Lundström[18] in 1962, and Tartakow[27] in 1965, provided a more accurate appraisal of the frequency of congenital defects. On the basis of these studies the over-all incidence of incapacitating congenital defects as a result of maternal rubella has been estimated at 10 per cent. In addition, these later studies also suggested that there is a definite epidemiologic factor involved—the incidence of congenital defects being higher during epidemics of rubella than in sporadic cases. The impact of the more recent studies is reflected in the following statements concerning the advisability of abortion. In 1947, Wesselhoeft[32] wrote; "The cumulated factual evidence presents a challenge to the existing law relative to therapeutic abortion. Although further investigations are needed to determine the accuracy of the evidence, it is certainly sufficient to warrant consideration of another amendment to grant permission for surgical interference after an attack of rubella."

In 1951, Swan[26] wrote: "On this basis (present statistics) it would appear ethical to subject every woman who contracts the disease during the first four months of pregnancy to therapeutic abortion."

In 1961, however, Pitt[20] wrote . . . "medical opinion now emphasizes that it is the duty of the doctors to treat the rubella-affected child, and not to terminate its life." In 1962, Lundström[18] in the same vein concluded . . . "maternal rubella *per se* does not constitute an indication for legal abortion and pregnancies should be terminated only when additional factors weigh heavily in the balance."

The recent preliminary communication by Rudolph *et al.*[23] prompted our interest in this subject. They reported additional findings in 25 infants, including unusual long bone changes, runting, purpura, thrombocytopenia and hepatosplenomegaly. In their experience the incidence of cardiac malformations and osseous changes exceeded 60 per cent in infants born of mothers who contracted rubella in the first trimester of pregnancy during the virulent epidemic in the spring and summer of 1964.

The affected newborn can harbor the viable virus up to 18 weeks of age. Thus, from a practical standpoint, it is important to make the diagnosis early. Precautionary measures can then be taken to prevent any unwitting exposure of pregnant hospital personnel to such an infant. The "measles

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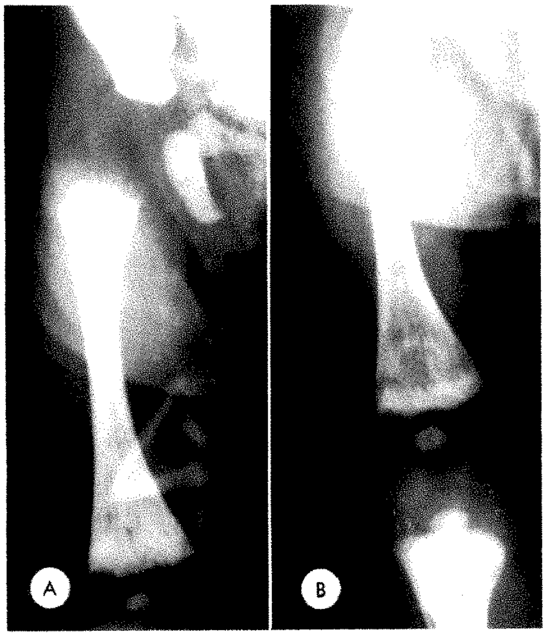

Fig. 1. (*A*) Alternating radiolucent and dense longitudinal metaphyseal striations produce a "celery-stick" appearance in the femoral metaphyses of this week old female with rubella embryopathy. Note failure of normal modelling and diminutive distal femoral ossification center. (*B*) At the age of 8 weeks, spontaneous healing of the metaphyseal striations is demonstrated. Abnormally dense texture and failure of metaphyseal tubulation indicate abnormal endochondral bone formation.

minded" radiologist may be afforded the opportunity to provide the first hint of the likely diagnosis.

Because of the current widespread epidemic of rubella in this country and the frequency of associated roentgenologic findings, it is imperative that every radiologist be familiar with the roentgenologic features of rubella embryopathy. This paper deals with 7 such recently encountered cases.

SKELETAL FINDINGS

Bone involvement was reported in 60 per cent of infants with congenital rubella syndrome by Rudolph *et al.*[23] The skeletal findings in our cases varied from minimal to marked. The principal changes occurred in the metaphyses of the long bones, particularly the distal femurs, and consisted of alternating dense and radiolucent longitudinal striations (Fig. 1*A*). These produce a celery-stick appearance in the affected metaphyses. Similar, but less marked, changes were noted in the anterior ends of the ribs in 1 patient. Failure of the normal modelling process resulted in flaring of the involved metaphyses. In 1 of our cases it produced generalized enlargement of the ribs and clavicles. Skeletal retardation may be manifested by absent or diminutive knee ossification centers at birth in full term infants and is probably secondary to the generalized growth retardation reportedly present in 80 per cent of cases.[23] Periosteal proliferation was not observed in any of the affected long bones.

Microcephaly occurs in some cases with mental retardation and may not be apparent in the first months of life. The pneumoencephalogram obtained in 1 of our infants with microcephaly and neurologic deficit demonstrated generalized cerebral atrophy (Fig. 2). Although this case has not been followed for a long period by serial skull roentgenograms, premature closure of the sutures would be an anticipated sequela.

The skeletal lesions healed spontaneously with a disappearance of the longitudinal metaphyseal striations. Irregular areas of sclerosis, however, persisted in the previously involved metaphyses for a variable period (Fig. 1*B*). As growth occurred these dense areas migrated toward the diaphysis but remained visible. In 1 newborn infant only minimal residual striations were present in the more severely affected metaphyses, while sclerosis was already present in other metaphyses, indicating that healing in them had already begun prior to birth.

We have not observed identical skeletal lesions in any other condition and believe that in most cases the combination of the characteristic bone findings and the history of maternal rubella should dictate the diagnosis of rubella embryopathy. In atypical cases or where the history of maternal rubella is uncertain, appropriate further studies may be necessary to exclude congenital syphilis, hypophospha-

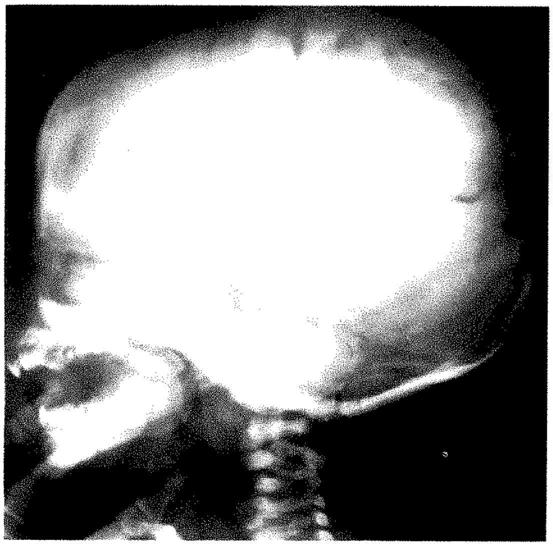

FIG. 2. Pneumoencephalogram of the infant shown in Figure 1, *A* and *B* shows cerebral hypoplasia and a mild degree of microcephaly.

tasia, metaphyseal dysostosis and rickets, although it is emphasized that seldom should any of these diseases pose a difficult problem in differentiation from rubella osteopathy.

CARDIOVASCULAR FINDINGS

The cardiac malformations in 3 of the patients (aged 2 weeks, 1 month and 4 years respectively) were defined by angiocardiography. The diagnostic approach in each case was similar. Under local anesthesia a transverse incision was made in the right cubital fossa and the right brachial artery was isolated immediately beneath the biceps expansion. The artery was opened and the tip of an angiocardiographic catheter was inserted and advanced into the ascending aorta. Contrast material was injected and films were exposed in two planes at the rate of 8, 4, 4, 2, 2 per second for 5 seconds. Following this, the catheter was removed and the opening in the brachial artery was closed with 6-0 vascular silk.

In 1 patient, following the aortogram, the tip of the catheter was advanced into the left ventricle and contrast material was injected to investigate the possibility of an associated ventricular septal defect.

Each patient showed the presence of a patent ductus arteriosus. In addition, 1 patient showed a stenosis of the left pulmonary artery at its origin and 2 patients exhibited pulmonary insufficiency.

PULMONARY FINDINGS

One of the 7 infants studied had diffuse bilateral interstitial infiltrates which had progressed slightly over a 2 month period (Fig. 5). Despite marked respiratory distress, when removed from oxygen, the baby had no auscultatory findings. Because Pneumocystis Carinii was suspected clinically and roentgenologically, lung biopsy was performed. This revealed an interstitial infiltrate consistent with viral pneumonitis. In the 6 weeks since lung biopsy, the respiratory distress has markedly decreased and the pulmonary infiltrates have slowly but incompletely resolved. The osteopathy of the femurs and tibias (Fig. 6, *A* and *B*) also present in this infant showed signs of healing concomitantly.

DISCUSSION

Inasmuch as the metaphyseal long bone changes are rather characteristic and measles epidemics are common, the question arises as to why the above association has not been previously noted. While possible, it seems unlikely that such unusual skeletal changes would not have been observed by roentgenologists or, if observed, mistakenly relegated to some other category. The frequency of the associated skeletal lesions during the present rubella epidemic may indicate that the causative virus is of a more virulent type than usually encountered and, therefore, productive of an unusually severe form of rubella. Rapid passage of a virus through experimental animals increases its virulence. It is possible that accelerated transmission of the rubella virus in the current epidemic of almost pandemic proportions has evolved an unusually virulent strain.

The mechanism responsible for the skeletal changes is unknown and several possibilities exist. Transplacental transmission of the virus is known to occur[23] and direct invasion of the skeletal system could produce the roentgenologic findings. However, the infrequency of viral infections of bone, the lack of periosteal proliferation, the absence of any systemic manifestations of osteomyelitis and the spontaneous healing make this explanation unlikely. The most feasible causative mechanism is an alteration in the normal endochondral bone formation; this is probably a reflection of the generalized retardation which is pres-

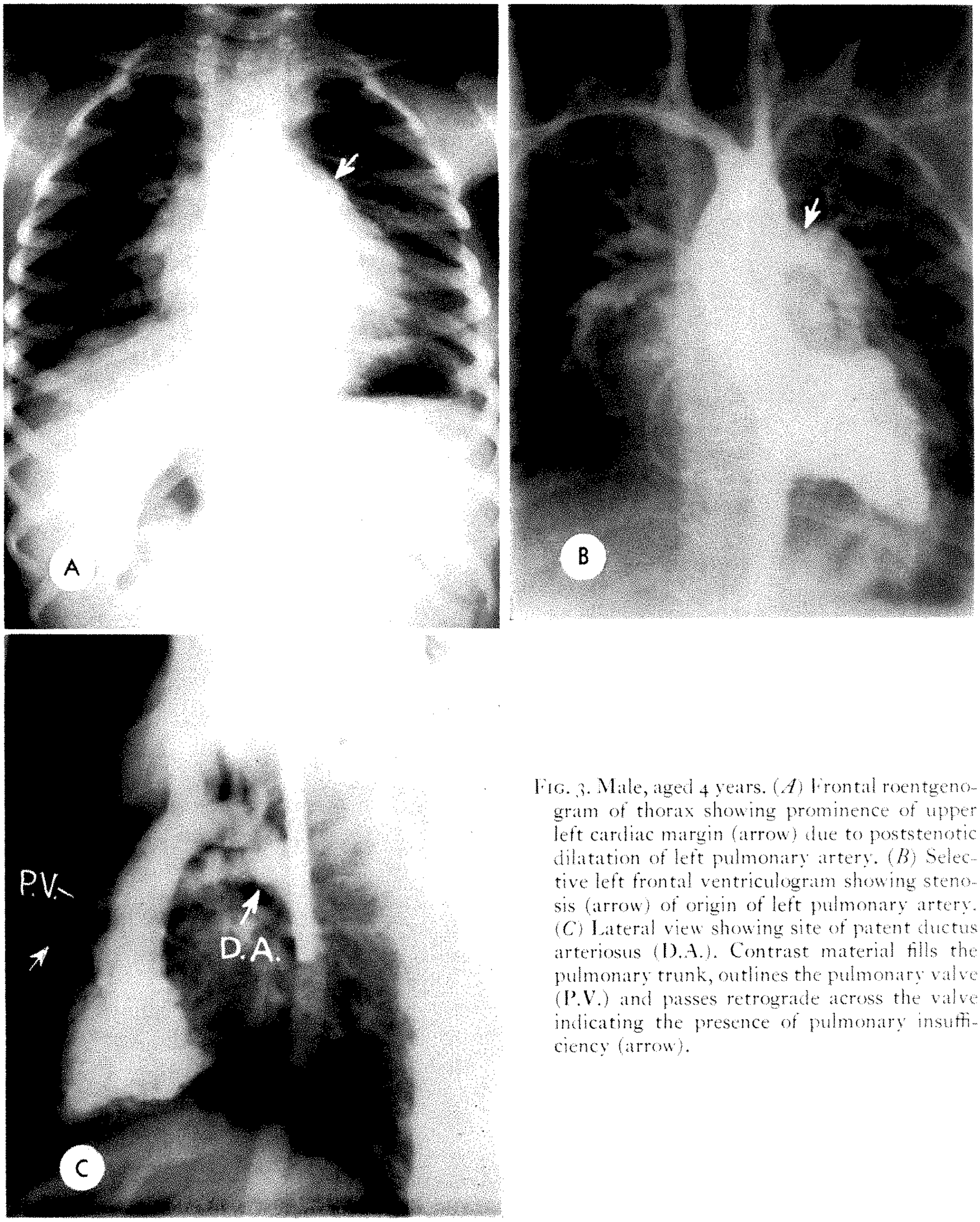

Fig. 3. Male, aged 4 years. (*A*) Frontal roentgenogram of thorax showing prominence of upper left cardiac margin (arrow) due to poststenotic dilatation of left pulmonary artery. (*B*) Selective left frontal ventriculogram showing stenosis (arrow) of origin of left pulmonary artery. (*C*) Lateral view showing site of patent ductus arteriosus (D.A.). Contrast material fills the pulmonary trunk, outlines the pulmonary valve (P.V.) and passes retrograde across the valve indicating the presence of pulmonary insufficiency (arrow).

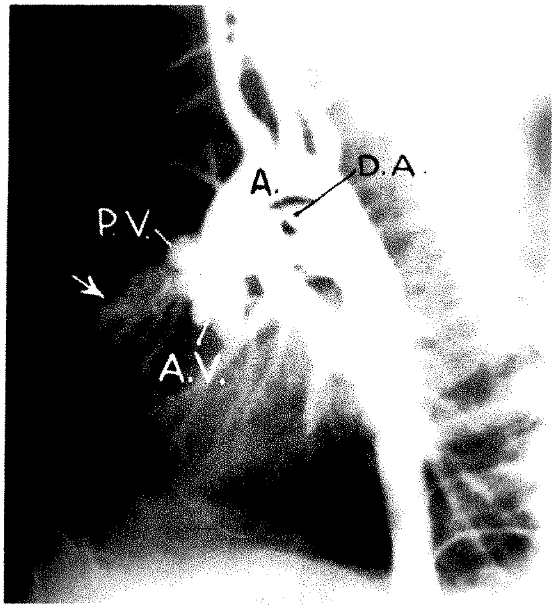

FIG. 4. Male, aged 1 month. Selective lateral thoracic aortogram showing patent ductus arteriosus (D.A.) and pulmonary insufficiency (arrow). P.V. = pulmonary valve; A.V. = aortic valve; A. = aorta.

ent and also responsible for the runting found in many of these infants. The conclusion of Engeset *et al.*[9] that the principal bone changes in infantile syphilis were due to deviations in the normal growth process rather than to the formation of specific granulation tissue in the affected bones supports this concept.

Inasmuch as the rubella virus has been cultured from the products of conception,[1] it is conceivable that viral placentitis could occur and result in placental insufficiency with secondary growth retardation, so prevalent in these infants. The rapid and spontaneous healing of skeletal lesions in the postnatal period, when nutrition is no longer dependent on the placenta, is supportive evidence for this etiologic possibility.

Rubella virus is recoverable from the throat washings and urine of infants with rubella embryopathy at birth and for varying periods thereafter.[1] The development of typical rubella in 1 of the nurses caring for an infant with rubella embryopathy[23] indicates that some of these affected infants are capable of transmitting the virus in viable form. Isolation of such an infant from pregnant personnel in the obstetrical and nursery departments is thus advisable. Similarly, it is also advisable to isolate the infant born to the mother with a classic history of rubella during the first trimester of pregnancy. However, some cases of rubella are clinically atypical and in mild form may be difficult to distinguish from the exanthemas secondary to other causes, including infectious mononucleosis, Echo and Coxackie viruses. Until such a time as the recently developed diagnostic test for rubella infection becomes generally available, it remains impossible to determine with certainty whether the rubella virus is responsible for the clinical findings of rash, fever and lymphadenopathy in some women in the first trimester of pregnancy. The slight fetal risk entailed in obtaining a roentgenogram of the maternal abdomen during the last month of pregnancy may be more than offset by the potential value of demonstrating the skeletal lesions of

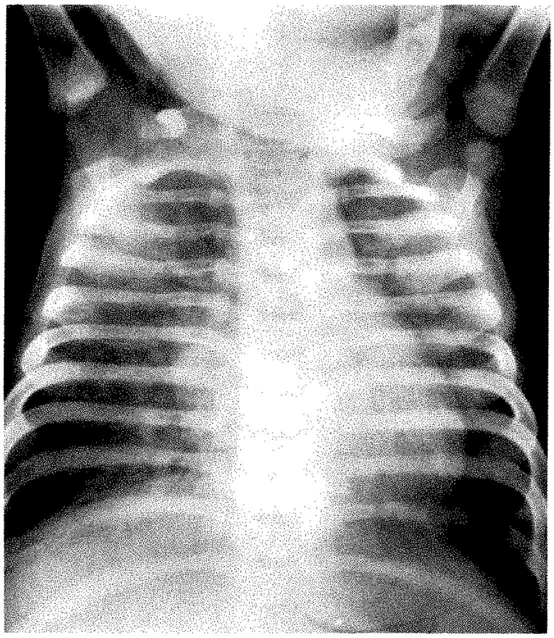

FIG. 5. Male, aged 3 months. Slowly progressive, diffuse interstitial pneumonitis proven by lung biopsy. Note healed phase of rubella osteopathy in both upper humeral metaphyses. Anterior ends of the ribs demonstrate lack of tubulation.

rubella in an affected fetus. Such positive knowledge prior to delivery would enable proper advance precautions to be taken.

The high incidence of cardiac malformations in newborn infants with rubella embryopathy is well documented. The most common anomalies encountered are patent ductus arteriosus and/or pulmonary stenosis, either at the level of the pulmonary valve or within the pulmonary trunk and its branches. Gibson and Lewis[12] described 17 cases of rubella cardiac malformations of which 14 showed a patent ductus arteriosus. Rowe[22] reported 31 infants with rubella cardiac anomalies. Twenty-two had a patent ductus arteriosus either as an isolated lesion (in 16) or in association with an additional cardiac malformation (in 6). Seven infants were shown to have stenosis of the central and/or peripheral pulmonary vessels. Emmanouilides *et al.*[8] demonstrated the presence of unilateral or bilateral pulmonary artery stenosis in 9 of 14 patients with a patent ductus arteriosus and a history of maternal rubella. Five of the 14 patients also showed an associated stenosis of the pulmonary valve. Three of our patients were studied by angiocardiography and each showed a patent ductus arteriosus. In addition, 1 infant had an associated stenosis of the origin of the left pulmonary artery (Fig. 3, *A*, *B* and *C*). Two patients showed the presence of pulmonary valvular insufficiency (Fig. 4). Collective experience in our community suggests that pulmonary valvular insufficiency is more common than hitherto suspected and may be demonstrated by selective thoracic aortography in the presence of a patent ductus arteriosus. When contrast material is delivered selectively by a catheter tip positioned in the ascending aorta, films exposed at the rate of 8/sec. during the first second will usually show clear definition of the ductus and the pulmonary trunk and its branches. In this way, the pulmonary vessels may be evaluated for the presence of stenotic lesions and any existing insufficiency of the pulmonary valve defined. In the 2 patients who showed pulmonary insufficiency, it is reasonable to assume that such insufficiency resulted from malformation of the pulmonary valve: the pulmonary trunk was of normal size and there was certainly no evidence to suggest that the insufficiency was due to dilatation of the pulmonary trunk and its valve ring, secondary to increased pulmonary flow through the patent ductus. Vlad *et al.*[31] following necropsy examination of 6 patients, aged 2 days to 14 years, with pulmonary regurgitation remarked, "it is probable that pulmonary regurgitation in infants is always due to either agenesis or a severely malformed pulmonary valve rather than to secondary dilatation of the valve." We might modify this statement and suggest that there are probably varying degrees of malformation of the pulmonary valve reflected by predominant insufficiency at one end of the spectrum and predominant stenosis at the other. To support the thesis that malforma-

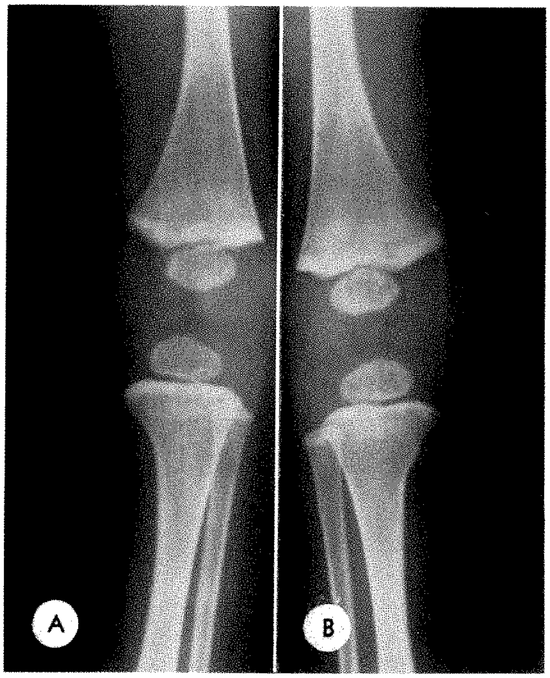

FIG. 6. (*A* and *B*) Same infant as shown in Figure 5 demonstrating undertubulation of the distal femurs and upper tibias. Note also the thickened, dense, irregular provisional zones of calcification, indicative of the healing stage of this disease.

tions of the pulmonary valve are relatively common in rubella cardiopathy, we reviewed 92 cases of pulmonary artery stenosis (trunk and branches) in the English language literature which were proven either by angiocardiography, surgery or necropsy.[2–12,14,15,17,21,22,24,28,29,30,38] In 22 patients a definite history of maternal rubella was recorded. In 5 other patients, no history of maternal rubella was mentioned, but it was noted that each of the 5 suffered from congenital cataract and/or deafness, strongly suggesting rubella embryopathy.

Of the 22 patients with definite rubella cardiopathy, pulmonary valvular stenosis was present in 13 and varied from mild to severe in degree. In addition, 14 of the 22 patients showed a patent ductus arteriosus.

Less commonly, other types of cardiac malformations, such as ventricular septal defect, atrial septal defect, coarctation of the aorta and aortic stenosis, occur following maternal rubella.

In view of the frequency of patent ductus arteriosus and pulmonary stenosis (valvular, trunk or branches) in rubella cardiopathy, we believe that selective thoracic aortography is the initial diagnostic procedure of choice unless there is some sound clinical evidence for the presence of one of the less frequently encountered cardiac malformations. Even at times when a septal defect is suspected in addition to a patent ductus arteriosus, selective thoracic aortography is still, in our opinion, the best initial procedure. When thoracic aortography is carefully and correctly performed, a good concentration of contrast material appears in the pulmonary vessels and, subsequently, clear definition of the left atrium and left ventricle will occur allowing detection of a left-to-right shunt at either the atrial or ventricular level, if present.

Little mention of interstitial pneumonitis as a component of the rubella syndrome has been made. Korones *et al.*[16] reported roentgenologic evidence of pneumonitis in 6 of 17 infants studied. The morphologic findings of interstitial pneumonitis were demonstrated in 2 of these babies at autopsy and the virus was isolated from lung sections in each case.

SUMMARY

1. The roentgenologic features of rubella embryopathy, with particular emphasis on the skeletal and cardiac findings are presented and their significance is discussed.

2. The role of selective thoracic aortography as the initial diagnostic procedure in delineating the commonly encountered cardiovascular malformations is outlined.

3. The roentgenologist should be aware of the roentgenologic manifestations of the rubella syndrome, inasmuch as he may be afforded the first opportunity to suggest the diagnosis.

4. Early diagnosis is essential for the protection of pregnant hospital personnel, since the affected infant is able to harbor and transmit the rubella virus.

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ROENTGEN APPEARANCE OF ANOMALIES ASSOCIATED WITH HYPOPLASTIC ANEMIAS OF CHILDHOOD: FANCONI'S ANEMIA AND CONGENITAL HYPOPLASTIC ANEMIA (ERYTHROGENESIS IMPERFECTA)*

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ROENTGENOLOGY has contributed greatly to the diagnosis of certain blood dyscrasias. Such entities as sickle-cell anemia, Mediterranean anemia, leukemia, and hemophilia often present characteristic roentgen changes that are widely recognized. Two additional entities, Fanconi's anemia and erythrogenesis imperfecta, have not as yet been described in the radiologic literature. In both these diseases, a high incidence of congenital anomalies prevails, the roentgen appearance of which may often be of value in establishing the correct diagnosis.

FANCONI'S ANEMIA

Smith,[29] in 1919, reported aplastic anemia in a 6 year old boy, associated with marked nuchal, genital, umbilical, and areolar skin pigmentation. Fanconi,[16] in 1927, described a pernicious-like anemia which developed in 3 brothers between the ages of 5 and 7 years. All 3 had unusual pigmentation of the skin, microcephaly with mental retardation, and were of shorter than normal stature. Testicular hypoplasia was noted in 2 of the brothers. Pigmentation and testicular hypoplasia were associated with multiple renal and skeletal anomalies in a patient with pancytopenia reported by Uehlinger,[30] in 1929. Since that time, at least 90 additional cases of pancytopenia with associated congenital anomalies have been recorded,[25] most of them under the designation of Fanconi's anemia or Fanconi's syndrome. (The latter is entirely different from renal tubular insufficiency with cystinosis, also sometimes called Fanconi's syndrome.)

CLINICAL MANIFESTATIONS

The clinical manifestations of Fanconi's anemia may be grouped into 2 categories—those relating to the hematologic disturbance and those relating to the congenital anomalies. The present authors reviewed the reports in the literature of 68 patients with Fanconi's anemia. Although many of the external anomalies of this disease were recognizable at birth, the onset of the hematologic disturbance was invariably delayed. The youngest patient reported was 17 months and the oldest 22 years old when the anemia first became evident. Of these 68 patients, 90 per cent were under the age of 15 and the median age in this series was 7 years. A definite predilection for males was noted (43:25).

HEMATOLOGIC DISORDERS

The initial symptoms of the hematologic disturbance are often those related to anemia, such as pallor and fatigability. In many instances, however, a tendency to increased bleeding, manifested by easy bruising, epistaxis, or gastrointestinal bleeding, is the presenting complaint. Frequently, the initial clinical manifestations of the blood dyscrasia become apparent with an associated acute febrile illness, such as respiratory tract infections or measles.

Peripheral blood studies in the fully developed hematologic disturbance typi-

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cally reveal anemia, leukopenia, and thrombocytopenia. In earlier stages of the disease, deficiency of only one or two blood elements may be apparent. The anemia is typically macrocytic and hyperchromic; the leukopenia is characterized by a reduction in granulocytes with a relative lymphocytosis. The bone marrow reveals various degrees of degeneration or complete atrophy and hypoplasia, or aplasia of all cellular elements.

Usually the disease progresses slowly, in most patients terminating fatally with hemorrhage and sepsis within 5 years after the onset.

Nilsson[25] noted that 30 families accounted for 62 of the 82 patients included in his comprehensive review. Consanguinity was recorded between the parents in 6 of the affected families. Various phenotypic expressions of the genetic defect have been recorded. The hematologic disorder has occurred in siblings of patients with the fully developed syndrome, unassociated with congenital anomalies.[24] On the other hand, congenital malformations in the absence of hematologic disturbance have been noted in affected families.[22] An increased incidence of leukemia in families of affected children has been noted.[17] The mode of transmission of Fanconi's anemia has generally been considered to be autosomal recessive.[2]

Estren and Dameshek[14] reported 8 cases of familial hypoplastic anemia, unassociated with other congenital anomalies. Several authors[5,12,26] have reported cases of thrombocytopenia with associated aplasia of the radii. A possible relationship between these entities and Fanconi's anemia has been suggested.[18]

Lewis *et al.*[23] have reported 18 cases of skeletal anomalies associated with congenital heart disease. The published reproductions of roentgenograms of the hands in this report are remarkably similar to those seen in Fanconi's anemia, with skeletal anomalies limited to the upper extremities, particularly the thumb. The changes were predominantly on the radial aspect of the extremities. No hematologic disturbances were noted in their patients.

Table I

CONGENITAL ANOMALIES WITH FANCONI'S ANEMIA REPORTED IN 68 PATIENTS

	No.	Per Cent
Abnormal pigmentation	50	74
Skeletal anomalies	46	68
Retarded growth	37	54
Hypogonadism	27	40
Small head	19	28
Renal anomalies	20	29
Microphthalmia	13	19
Strabismus	11	16
Hyperreflexia	11	16
Undescended testis	7	16*
Deafness	4	6
Anomaly of external ears	3	4
Adrenal absent or hypoplastic	3	4
Inguinal hernia	3	4
Obesity	3	4
Hypospadias	2	5*
Hydrocephalus	2	3
Gynecomastia	2	3
Septate bladder, blue sclerae, anal stenosis, sacrococcygeal sinus, Meckel's diverticulum, and aqueductal stenosis with absent septum pellucidum	1 of each	

* Per cent of 43 males.

ASSOCIATED MALFORMATIONS

The congenital anomalies noted in the 68 patients reviewed are listed in Table I. Less obvious abnormalities may have been overlooked or may not have been sought in many instances. Hence, the figures do not necessarily represent the true incidence of congenital anomalies in this syndrome.

Abnormal Pigmentation. In every series reported, abnormal pigmentation was the most consistent abnormality observed. Although any area of the body could be involved, certain areas showed increased pigmentation more often. These were the trunk, the skin folds, such as the axillae and groins, and the area around the neck. Frequently, the pigmentation was apparent before onset of the pancytopenia; how-

Table II

SKELETAL DEFORMITIES IN 44 PATIENTS WITH FANCONI'S ANEMIA

Absent, hypoplastic, or supernumerary thumb	34
Hypoplastic or absent radius	9
Congenital hip dislocation	4
Webbing, second and third toes	4
Flat feet	2
Klippel-Feil deformity	2
Club foot	1
Extra terminal phalanges of third and fourth toes	1
Sprengel's deformity	1

ever, it sometimes appeared at a later stage in the disease. Histologic examinations have shown that the skin pigmentation is the result of abnormally increased melanin deposits.[21]

Skeletal Anomalies. The most frequent site of skeletal anomalies of Fanconi's anemia is in the upper extremities, particularly the thumb, and, less commonly, the first metacarpal and adjacent carpals. No specific pattern of association among the various anomalies could be ascertained, nor was there a particular tendency toward symmetric changes. Hypoplasia of the phalanges of the thumb was noted in 12 instances, complete absence of one or both thumbs in 15, and a supernumerary thumb in 3. The hypoplasia of the involved phalanges was usually manifested by both shortening and narrowing. Some phalanges, however, were of normal length but abnormally slender. Both the proximal and distal phalanges were hypoplastic in most instances; in a few, the distal phalanx alone was shortened. In most instances when hypoplasia of the phalanges of the thumb was present, shortening or thinning of the first metacarpal also was observed. Complete absence of the thumb was invariably associated with aplasia of the corresponding metacarpal. Hypoplasia of the first metacarpal with normal first phalanges has also been reported.[28]

Other abnormalities of the thumb have been described. These include atrophy of the thenar eminence, immobility of one or both joints of the thumb, and a double distal phalanx. In 3 patients, one or both thumbs were attached to the hand only by soft tissue.

Several authors have noted incurving fifth fingers, similar to those observed in mongolism, associated with hypoplastic middle phalanges of the fifth digit.[21] In several instances hypoplasia of the second and fifth middle phalanges was evident in published roentgenologic reproductions, although not described in the text.[9,18]

Unilateral absence of the radius was noted in 3 instances and bilateral absence in an additional 3. In each patient with radial aplasia, the corresponding thumb was also absent. In those reports that included reproduced roentgenograms, radial aplasia was associated with shortening and medial concavity of the ulna, the wrist being held in valgus position.

Other congenital malformations that were noted in the skeletal system in Fanconi's anemia are listed in Table II.

Renal Anomalies. More than one-fourth of the 66 patients reviewed had renal anomalies. In 14, absence of one kidney was associated with either renal hyperplasia or fusion on the opposite side. (Nilsson's[25] series included 3 horseshoe kidneys.) Simple ectopy was evident in 7 patients. In several instances, hydronephrosis or impaired excretion was evident on excretory urography.

Cardiovascular Anomalies. An abnormality involving the cardiovascular system was noted in 5 patients. A hypoplastic aorta with coarctation[8] in one instance and a patent ductus arteriosus[22] in another were found at necropsy. In 2 patients,[16,21] definite evidence of congenital heart disease was noted clinically, although not proved surgically or by necropsy. Marked atheromatous changes were found in the aorta of a girl who died at the age of 16 years.[28]

Other Anomalies. A wide range of other congenital deformities have been reported in association with Fanconi's anemia (Ta-

Fig. 1. Case 1. Fanconi's anemia in an 8 year old girl. The fifth middle phalanx is notched on its radial side distally. This phalanx is shorter on its radial aspect than on its ulnar, resulting in an incurving of the fifth finger (clinodactyly). The navicular is hypoplastic.

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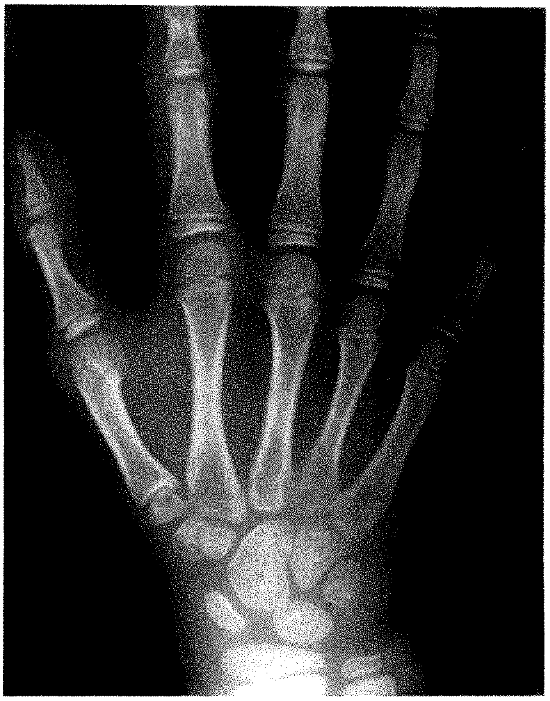

ble 1). Hypogonadism was found in both sexes. In males, this was most often manifested by testicular hypoplasia with underdeveloped external genitalia. Hypoplastic ovaries, infantile uterus, and atresia of the cervix and vagina were the manifestations in the females.

The degree of growth retardation in the patients reported was mild to moderate. None of the patients showed the extreme degree of dwarfism of osteochondrodystrophy. Similarly, the microcephaly did not approach the extreme degree noted in association with severe congenital anomalies of the brain. The microcephaly in most patients was manifested by a head circumference of a few centimeters less than normal. Microphthalmia was present in some cases but also was often of minimal degree.

Cole, Cole, and Lascheid[7], and Bryan and

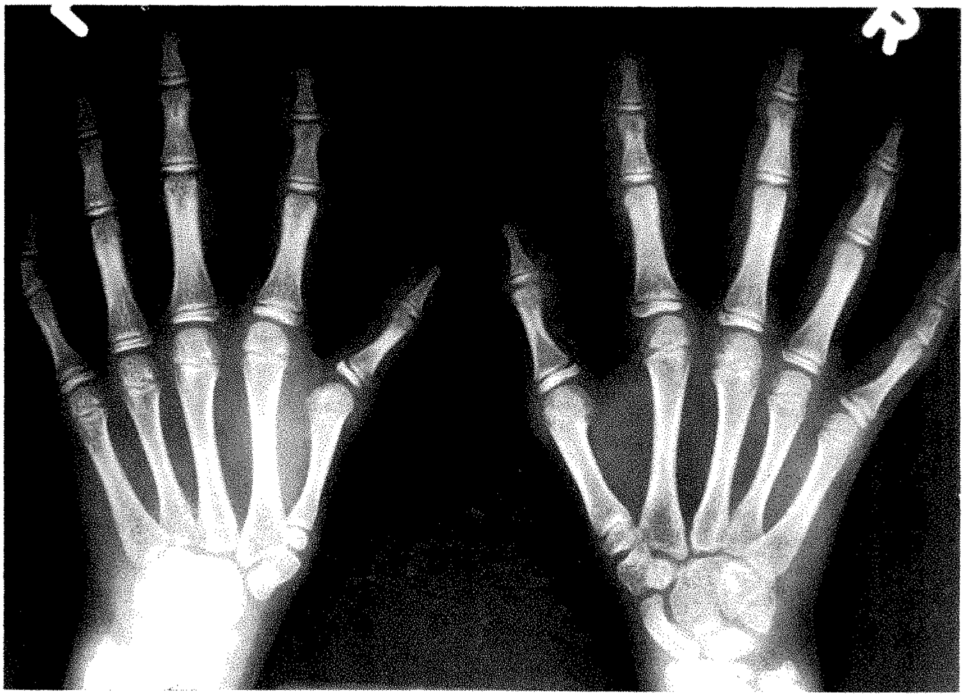

Fig. 2. Case 1. At the age of 13 years, the left first metacarpal is deficient proximally and the phalanges of the thumb are hypoplastic. Clinodactyly of the left fifth finger is more pronounced than on the right. The left navicular is hypoplastic. Although the right navicular at the age of 8 years (Fig. 1) was somewhat smaller than normal, at 13 years it is unusually long.

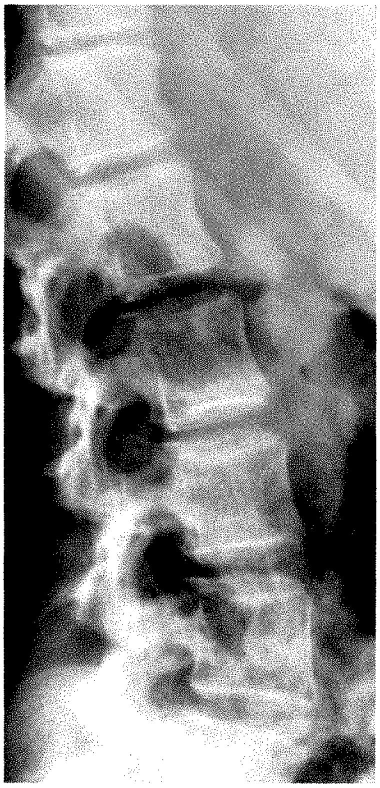

Fig. 3. Case I. The lumbar vertebral bodies are somewhat narrow in their anteroposterior dimension, suggesting the appearance of "caninization."

Nixon[3] have called attention to a possible relationship between Fanconi's anemia and dyskeratosis congenita. They suggested that the skin disorder is a further variant of the diverse congenital defects associated with Fanconi's anemia.

We have had the opportunity of reviewing the roentgenograms of the following 4 patients, in whom Fanconi's anemia was diagnosed.

REPORT OF CASES

Case I. This 13 year old girl was first seen because of pancytopenia in 1959 at the age of 8 years. In addition to the hematologic disorder, a shorter than normal stature was evident. Multiple skeletal anomalies were present as were abnormal pigmentation and delayed sexual maturation. Roentgenologically, the hands were of primary interest (Fig. 1 and 2). The phalanges of both thumbs were short and slender, more marked on the left side. Both first metacarpals were short, that on the left appreciably narrow at its proximal end. The fifth middle phalanges were slightly notched on the inner aspect of their distal ends. They were shorter on their radial aspects than on their ulnar. This shortening resulted in clinodactyly of the fifth digits, similar to that seen in mongolism. Although the navicular of the right wrist had been smaller than normal at age 8 years, it was longer than normal at the age of 13 years. The left navicular was hypoplastic. Generally, the long bones were more slender than usually seen. The lumbar vertebral bodies were also unusually slender in their anteroposterior diameters, resembling the "caninization" described in patients with paralysis or muscular weakness (Fig. 3).

Bone surveys of both parents were obtained and showed no abnormalities. Two of the patient's 3 siblings, however, both girls, of 8 and 17 years, showed definite clinodactyly of the fifth fingers. A deformity of the middle

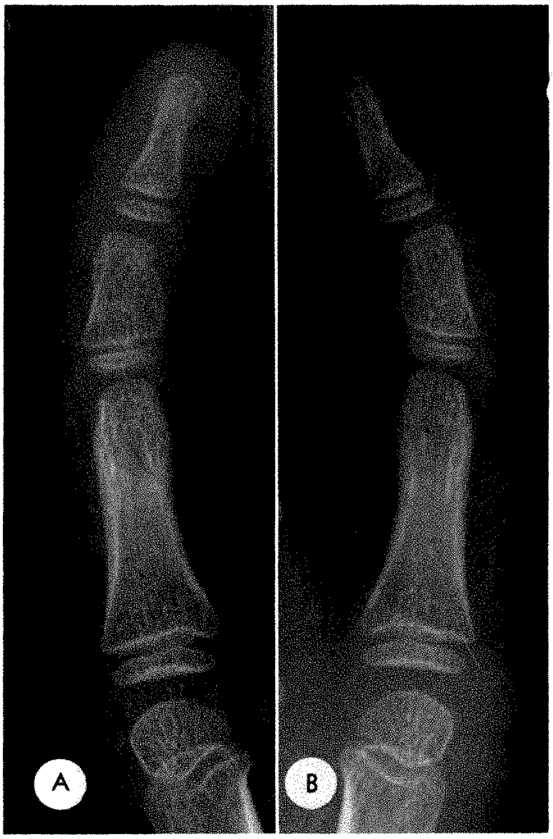

Fig. 4. (*A* and *B*) An 8 year old sister of Case I. The distal portion of the middle phalanx on its radial aspect is slightly notched, similar to that of the patient. No hematologic abnormality.

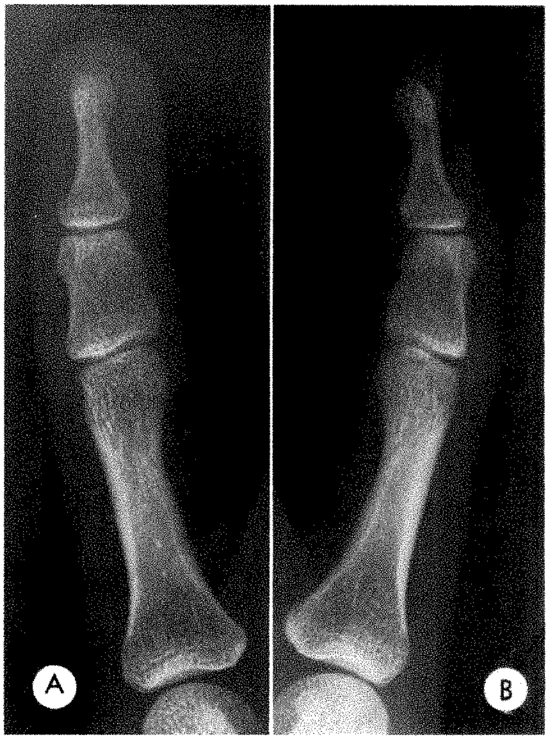

Fig. 5. (*A* and *B*) A 17 year old sister of Case I. Although the notching of the phalanx is less apparent in this girl than in her sisters, the radial side of the middle phalanx is shorter than the ulnar side, resulting in clinodactyly. No hematologic abnormality.

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phalanges similar to that of the patient was also present (Fig. 4, *A* and *B*; and 5, *A* and *B*). Similar changes, but of lesser degree, were seen in an 11 year old brother. In none of the siblings has evidence of hematologic abnormality been seen.

Case II. This 11 year old girl was previously reported by Silver, Blair, and Kempe.[27] In addition to pancytopenia and abnormal pigmentation, there were present microcephaly, microphthalmos, strabismus, dwarfism, mental retardation, and bilateral slight syndactylism of the second and third toes. Roentgenologically, the skull was smaller than normal in all dimensions. The sella turcica was of small size but

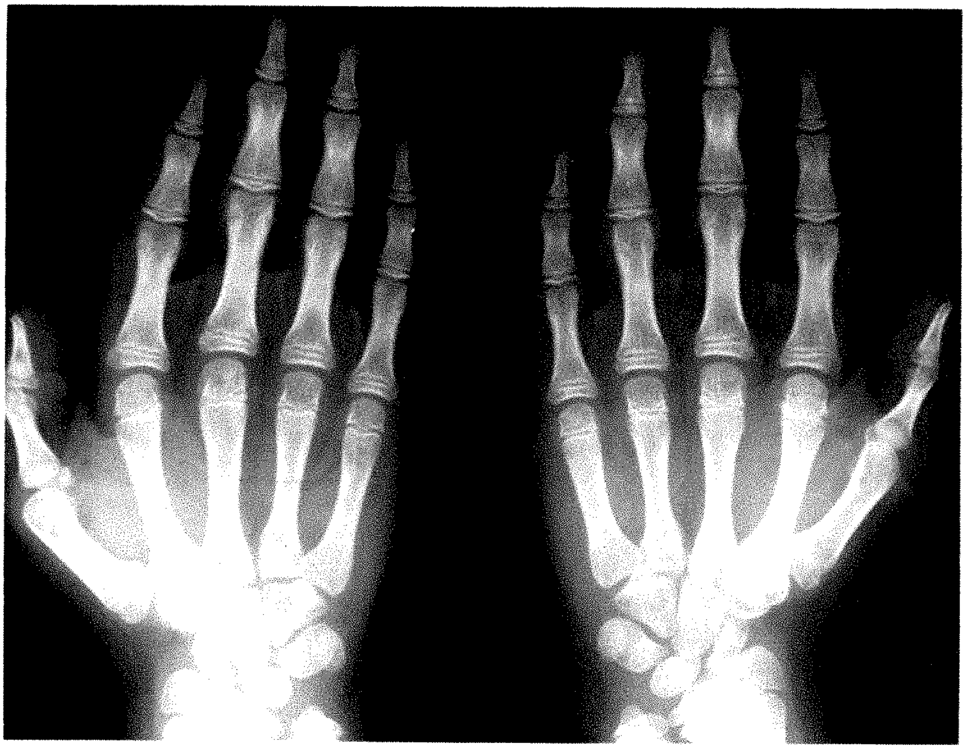

Fig. 6. Case II. An 11 year old girl with Fanconi's anemia. No bone abnormality. Fullness of the soft tissues of the fingers proximally gives the fingers a conical appearance. The tubular bones are short, but otherwise normal.

volume was within the normal range. Although, clinically, immobility of the interphalangeal joint of the right thumb was noted, no roentgenologic abnormality was apparent in this joint. The hands were short and broad with fullness of the soft tissues of the fingers proximally, giving the fingers a somewhat conical appearance (Fig. 6). The tubular bones of the hands were proportionately short. No abnormality was evident on excretory urography or on examination of the chest.

Case III. Associated with pancytopenia in this 6 year old boy were dwarfism, hypogonadism, and abnormal skin pigmentation.

Case IV. A younger sister of Case III was of short stature and, in addition to pigmentation and aplastic anemia, had strabismus. No evidence of skeletal anomalies was seen in either sibling, although the sister had bilateral rudimentary cervical ribs.

ERYTHROGENESIS IMPERFECTA

In 1938, Diamond and Blackfan[11] described 4 cases of a syndrome characterized by a slowly progressive anemia beginning early in infancy. Two similar cases had been described by Josephs[20] 2 years previously. In none of these patients had there been jaundice, leukopenia, or thrombocytopenia. The anemia had been the result of a selective hypoplasia of red cell precursors in the bone marrow. Since these reports, well over 100 have been added to the literature. Various synonyms have been applied to the syndrome, including congenital hypoplastic anemia, erythrogenesis imperfecta, pure red cell anemia, and congenital aregenerative anemia.

In patients with erythrogenesis imperfecta, examination of the peripheral blood reveals a normocytic, normochromic anemia, with normal numbers of other formed elements. The reticulocyte count is less than 2 per cent despite the severe anemia. In the bone marrow, a complete or almost complete absence of cells of the erythroid series is characteristic. White blood cell and platelet precursors appear in normal numbers.

The prognosis, although guarded, is less grave than in Fanconi's anemia, many children having survived for years with periodic transfusions. Remissions after the administration of steroids and spontaneous remissions, sometimes after years of transfusion therapy, have also been reported. With repeated transfusions over prolonged periods of time, the changes of transfusion hemosiderosis often become an important part of the clinical picture.

Benign thymoma has been described in association with red cell aplasia in adults;[6] however, erythrogenesis imperfecta, as discussed in the present report, is characterized by its appearance in infancy, and thymoma has not been noted in children with this disease.

Although the etiology of the disease is unknown, a disturbance in tryptophan metabolism has been suggested by Altman and Miller.[1] These investigators identified anthranilic acid, a metabolite formed by the breakdown of tryptophan, in the urine of 9 patients with this disease. Isolated examples of a familial incidence have been recorded.[4,10]

CONGENITAL ANOMALIES

Congenital anomalies have not been emphasized as part of this syndrome, but 1 of

Table III

CONGENITAL ANOMALIES WITH ERYTHROGENESIS IMPERFECTA REPORTED IN 74 PATIENTS

Retarded growth		10
Mental retardation		4
Congenital heart disease		3
Skeletal anomaly		3
Congenital defect of osseous development	1	
Extra phalanx of one thumb	1	
Extra thumb	1	
Renal anomaly		3
Bilateral double ureters with congenital hydronephrosis	1	
"Congenital dysplasia" of one kidney	1	
Absent left kidney and ureters	1	
Webbed neck		2
Blue sclerae		2
Malformed ear, inguinal hernia, cleft palate, ptosis		1 of each

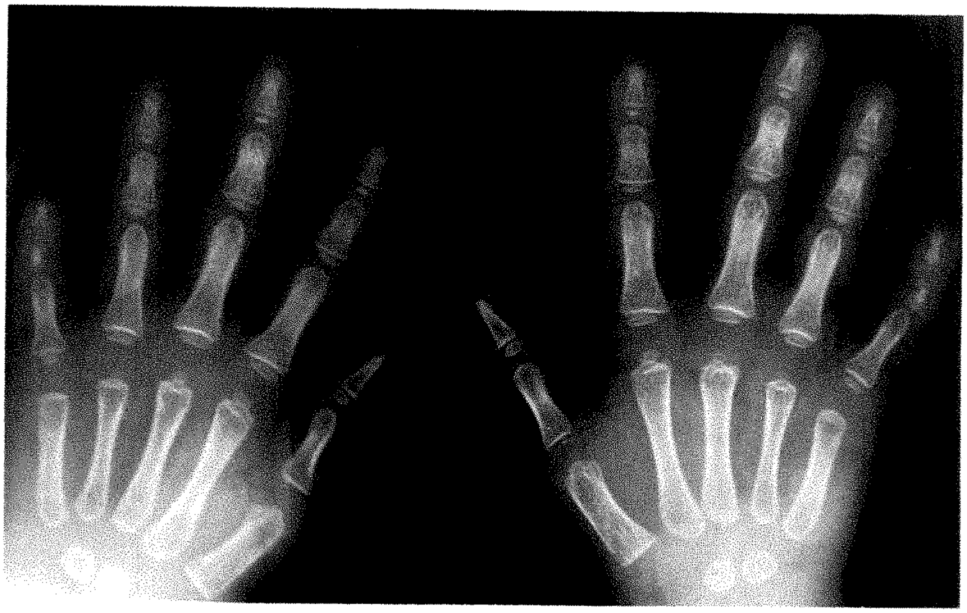

FIG. 7. Case v. Erythrogenesis imperfecta in a 31 month old girl. The bone age is that of an 18 month old girl. Although the ossification center of the distal radius is not included in this figure, it was unossified. Dysplasia of both fifth middle phalanges is evident.

our patients with this disease had such anomalies. These findings prompted a review of the literature to determine the frequency and type of anomalies in the patients previously reported. Case reports of 74 patients were reviewed, of which 40 were females and 34 males. Of these, 25, or 33.8 per cent, showed at least one developmental anomaly. Diamond, Allen, and Magill,[10] in a review of 30 of their own patients, found 7 with minor congenital anomalies. The congenital anomalies of the patients in our review are listed in Table III. The anomalies in erythrogenesis imperfecta are not as frequent, nor are the skeletal deformities as marked as in Fanconi's anemia. Nevertheless, the finding of maldevelopment in nearly one-third of the patients reviewed is more than can be accounted for by chance.

REPORT OF A CASE

CASE V. In this patient, a 4 year old girl, the results of the hematologic examination indicated erythrogenesis imperfecta. In addition, clinical and roentgenologic examinations and cardiac catheterization gave evidence of a ventricular septal defect. At the age of 6 weeks, the patient had undergone a surgical procedure for intrapulmonary sequestration. The sequestered lung had been receiving its blood supply from an aberrant vessel arising from the aorta. Other anomalies included clinodactyly of the fifth fingers and webbing of the fourth and fifth toes. Growth was retarded, height and weight being less than those of the third percentile. An additional finding of interest was the abnormal pigmentation of the skin which was described as almost bronze in color. Although anemic since the age of 3½ months, the patient had received only 2 transfusions; hemoglobin was being maintained at near normal levels with steroid therapy.

Roentgenologic examination of the hands (Fig. 7 and 8) at the age of 31 months demonstrated a bone age compatible with that of an 18 month old child. The ossification center of the distal radius was absent. Marked dysplasia of both fifth middle phalanges with resultant clinodactyly was evident. The short phalanges were unusual in that the center of the diaphyses was markedly narrow and the epiphysis absent. Examination of the chest disclosed cardiomegaly and pulmonary overcirculation, consistent with the diagnosis of ventricular septal defect. Also evident were the changes incident to prior removal of the pulmonary sequestration.

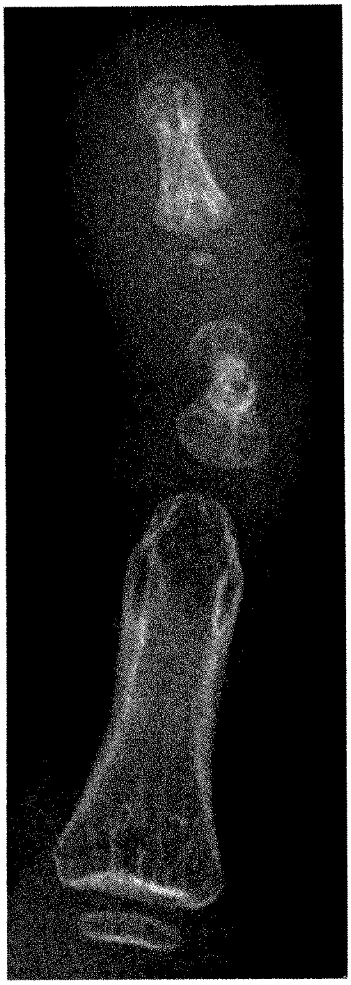

Fig. 8. Case v. Close-up view of fifth middle phalanx of Figure 7, demonstrating peculiar dysplastic charge.

A comparison of the findings in Fanconi's anemia and erythrogenesis imperfecta is given in Table iv.

Table IV

Data obtained in a review of Fanconi's anemia and erythrogenesis imperfecta

	Fanconi's Anemia	Erythrogenesis Imperfecta
Total no. of patients	68	74
Male	43	34
Female	25	40
Age range at onset of anemia	17 mo. to 22 yr.	Birth to 18 mo.
Average age at onset	8 yr.	2½ mo.
Median age at onset	7 yr.	2 mo.
Per cent of patients with at least 1 anomaly demonstrated	97%	33%

SUMMARY

Fanconi's anemia is a hematologic disorder, often familial, characterized by pancytopenia and diverse congenital anomalies. The most frequent of these anomalies are abnormal skin pigmentation, skeletal anomalies, primarily of the thumb, growth retardation, and hypogonadism. Microcephaly, renal anomalies, microphthalmia, and strabismus also are frequently associated with this disease.

Erythrogenesis imperfecta, or congenital hypoplastic anemia, is characterized by a hematologic deficiency limited to the erythrocytic series. It usually begins within the first year of life. A review of the literature disclosed approximately one-third of these patients with some form of maldevelopment. Most of the abnormalities were mental and physical retardation, congenital heart disease, and skeletal anomalies.

A discussion of the roentgen features in these diseases is presented, together with the findings in 4 patients with Fanconi's anemia and 1 with erythrogenesis imperfecta.

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COEXISTENT POLYOSTOTIC FIBROUS DYSPLASIA AND EOSINOPHILIC GRANULOMA OF BONE*

A UNIQUE ASSOCIATION

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FIBROUS dysplasia of bone and histiocytosis X are distinct disease entities. Each presents a broad spectrum of clinical, histologic and roentgenographic manifestations. These range from the single lesion of fibrous dysplasia to polyostotic involvement on the one hand and from the solitary eosinophilic granuloma of bone to the fulminant Letterer-Siwe's disease on the other.

The association of the two disease entities in a single individual is unusual.

REPORT OF A CASE

An 8 year old white female was apparently well until 1 month prior to admission, at which time she developed progressive pleuritic pain. This was associated with low-grade fever and a weight loss of 3 pounds. A chest roentgenogram showed an osteolytic lesion in the right 6th rib.

During the past 3 years, she had sustained fractures of the right radius and 2 right metacarpals. These injuries resulted from minor trauma and healed uneventfully.

On admission, she was noted to be a well developed 8 year old girl; her height and weight were in the 60th and 45th percentile, respectively. Fine pubic and axillary hair and early breast development were noted. A pigmented oblong area, measuring 3.5 by 1 cm., was present on the posterior aspect of the lower neck. The right arm was 2 cm. longer than the left.

Routine laboratory data were within normal limits, as were the urine output and tests of renal concentration and dilution. The serum calcium was 10.4 mg. per cent; the serum phosphorus was 4.6 mg. per cent, and the alkaline phosphatase was 16 and 13.8 Bodansky units on two determinations. The vaginal smear revealed a moderate estrogen effect.

Roentgenographic examination of the chest disclosed a 2 cm. defect in the right 6th rib surrounded by an extrapleural mass (Fig. 1).

A bone survey demonstrated multiple, sclerotic and radiolucent areas encroaching on the cortex from within and irregular widening of the medullary canal (Fig. 2, *A*, *B* and *C*; 3, *A* and *B*). There was expansion of some metacarpals, metatarsals and phalanges.

Overgrowth and sclerosis of bone were present in the right frontal area, the right maxilla, the occiput and the base of the skull (Fig. 4, *A*, *B* and *C*).

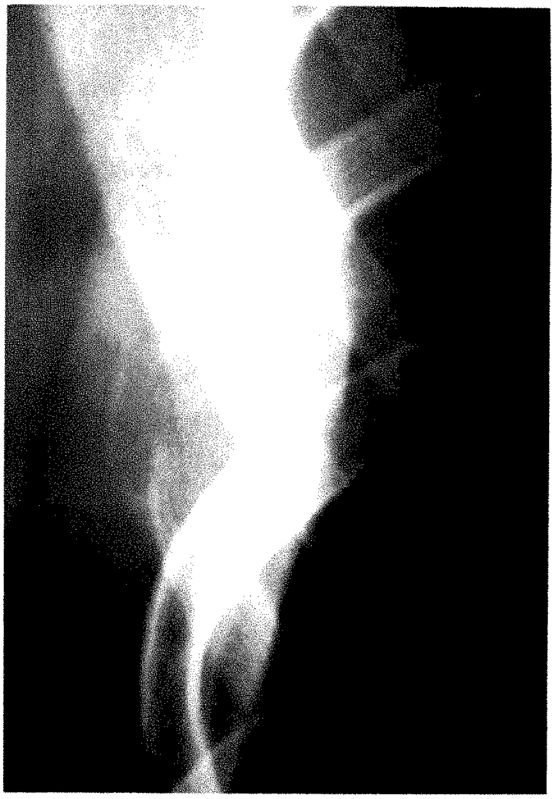

FIG. 1. Anteroposterior roentgenogram of the involved right ribs (magnified view) showing a sharply marginated defect in the mid-axillary line of the right 6th rib. Note underlying extrapleural mass.

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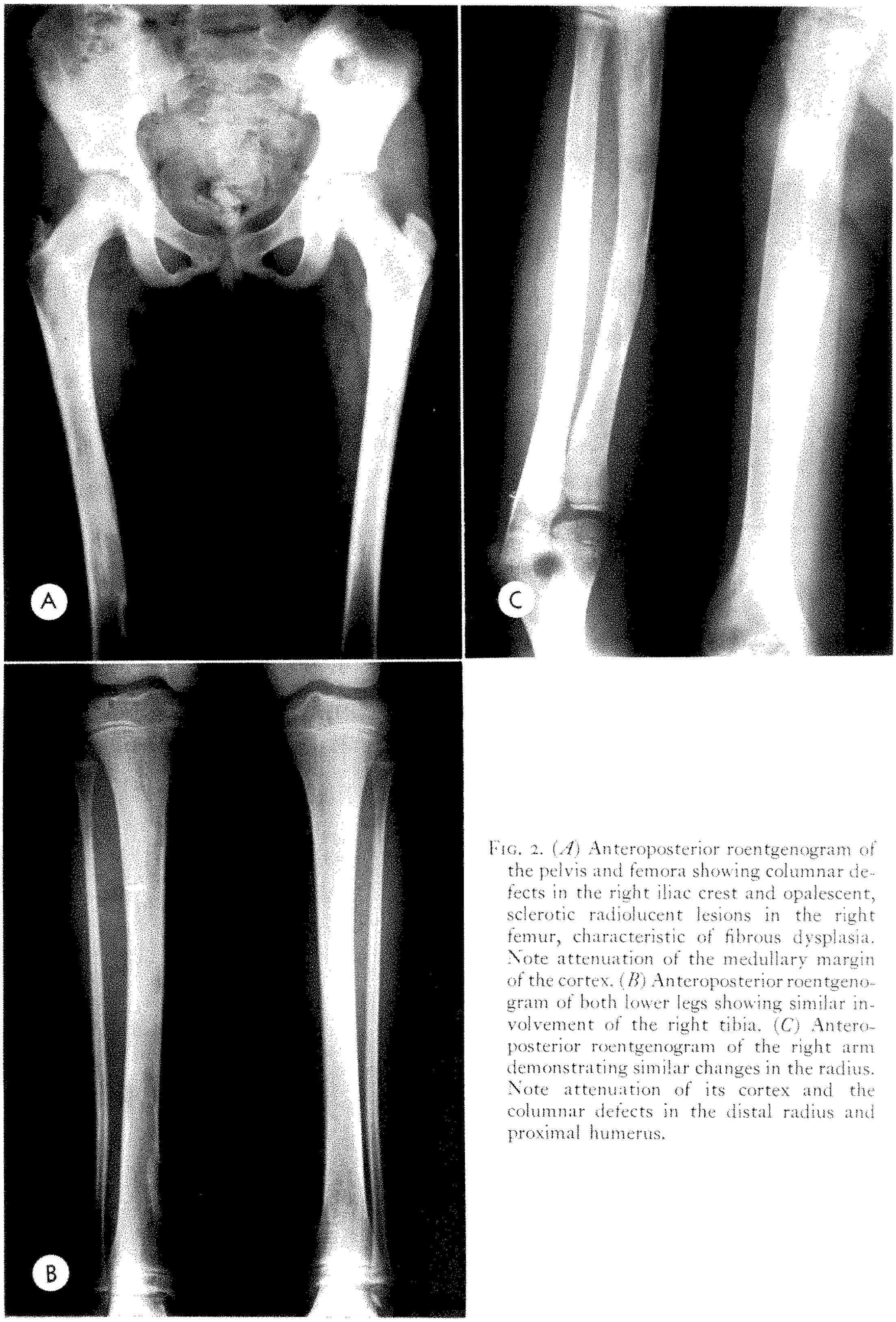

Fig. 2. (*A*) Anteroposterior roentgenogram of the pelvis and femora showing columnar defects in the right iliac crest and opalescent, sclerotic radiolucent lesions in the right femur, characteristic of fibrous dysplasia. Note attenuation of the medullary margin of the cortex. (*B*) Anteroposterior roentgenogram of both lower legs showing similar involvement of the right tibia. (*C*) Anteroposterior roentgenogram of the right arm demonstrating similar changes in the radius. Note attenuation of its cortex and the columnar defects in the distal radius and proximal humerus.

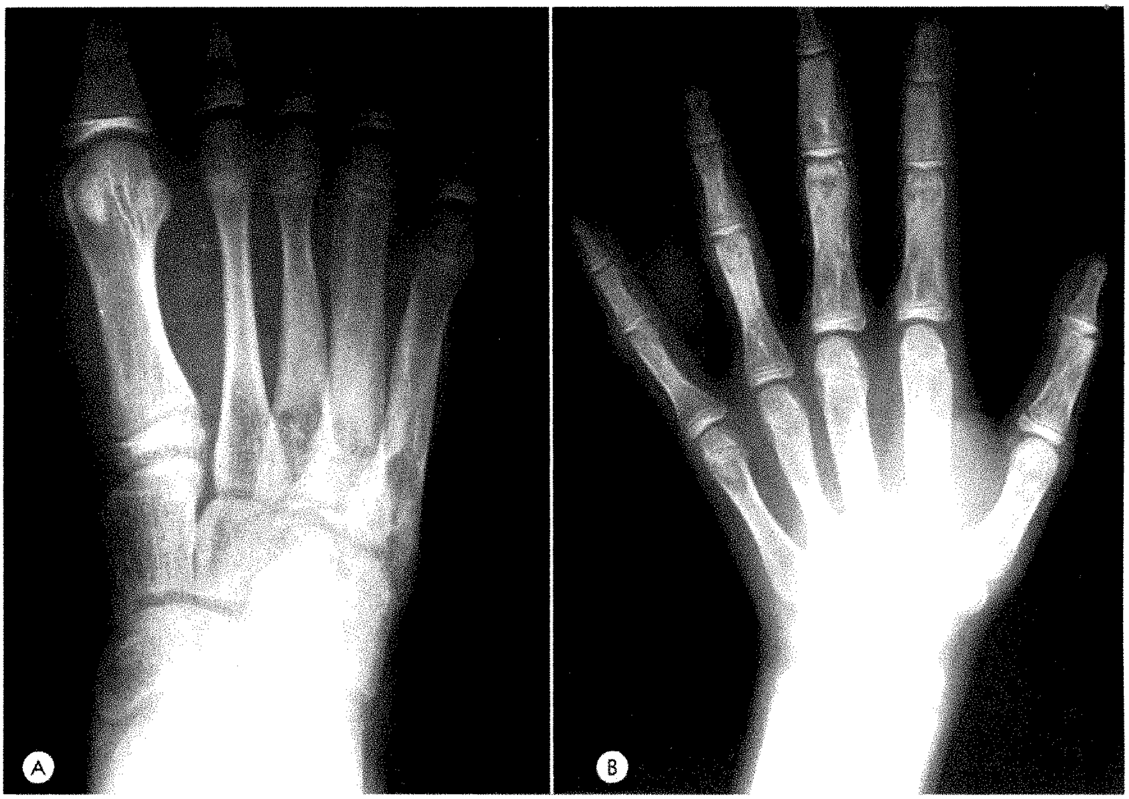

Fig. 3. (*A*) Anteroposterior roentgenogram of the right foot demonstrating columnar expansion of the third, fourth and fifth metatarsals; there is lack of normal trabecular pattern and a resulting diffuse ground-glass appearance. (*B*) Anteroposterior roentgenogram of the right hand demonstrating a similar involvement of the second and third metacarpals and their corresponding proximal phalanges.

With the exception of a small fibrous defect in the left first metacarpal, the osseous changes were entirely confined to the right side. All the bone lesions, excluding the destructive process in the rib, were considered to be characteristic of polyostotic fibrous dysplasia.

The bone age was 11 years as compared with the chronologic age of 8.

An excision biopsy of the lesion in the right sixth rib revealed numerous eosinophils, plasma cells, histiocytes, and rare multinucleated cells. Foamy cytoplasm or other evidence of intracellular lipid was not present, and there was no fibrosis. The lesion was typical of the proliferative phase of histiocystosis X (Fig. 5).

A wedge biopsy of the right iliac crest demonstrated the characteristic changes of fibrous dysplasia. There were areas of whorled cellular connective tissue in which thin, disoriented bone trabeculae and occasional osteoblasts were present (Fig. 6). Several islands of cartilage were encountered in the specimen but were without consistent relation to the fibrotic lesions. The epiphyseal plate showed irregularities in enchondral bone formation. It is probable that the islands of cartilage noted represent remnants of this growth plate irregularity (Fig. 7).

Four months following discharge from the hospital, the patient developed point tenderness over the left parietal area. Examination revealed a 3 cm. subcutaneous nodule which corresponded with the punched-out 0.5 cm. defect on the roentgenogram. No treatment was instituted, and, on re-examination 4 weeks later, the nodule was no longer palpable. Three months later, she sustained another fracture of the right radius following minor trauma.

Her first menstrual period occurred at the age of 9 years.

On follow-up examination 2 years later, complete regeneration of the excised portion of the right sixth rib had occurred, and there was complete healing of the defect in the left parietal bone. There was no visible change in the remaining unilateral skeletal lesions.

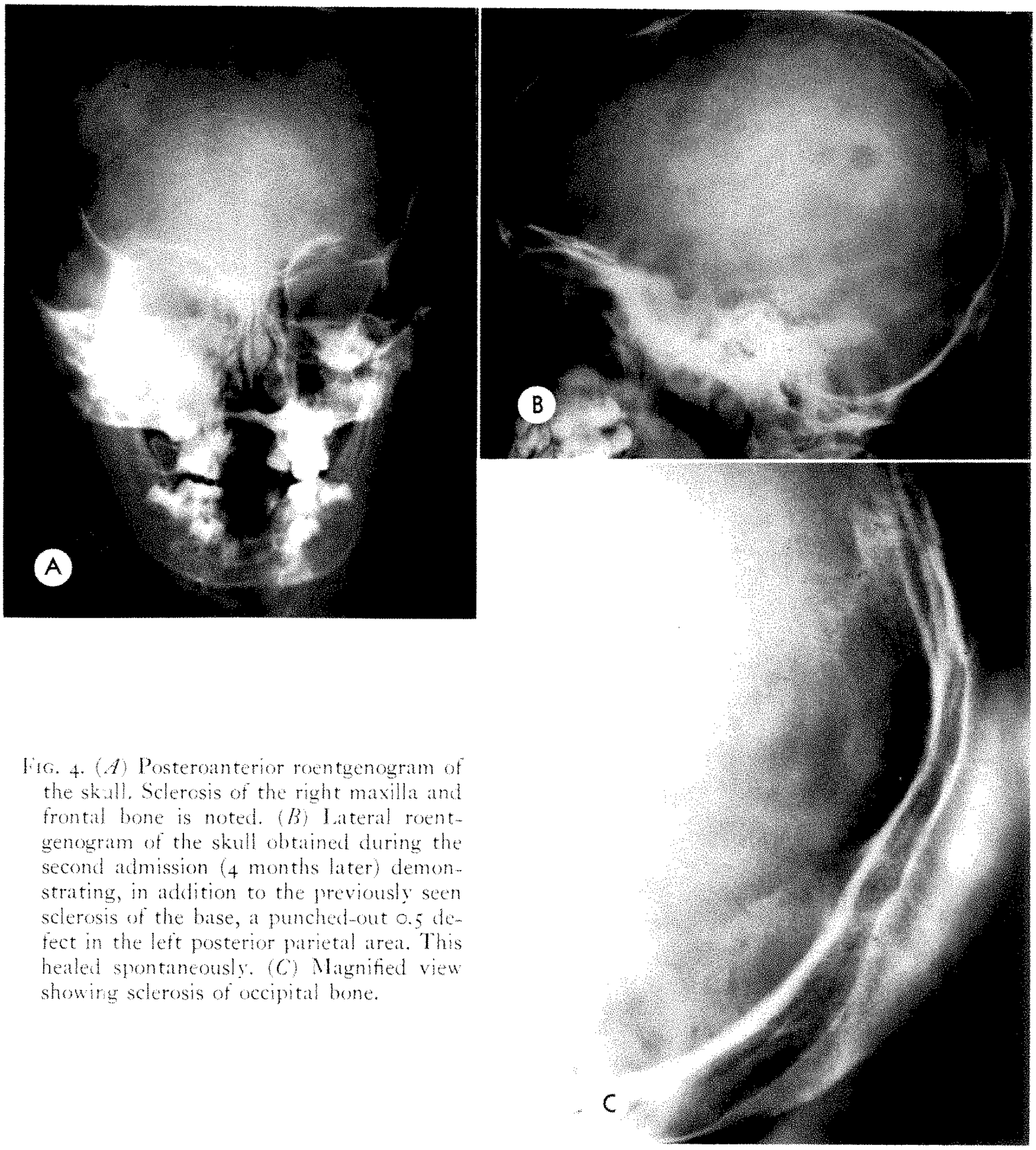

Fig. 4. (*A*) Posteroanterior roentgenogram of the skull. Sclerosis of the right maxilla and frontal bone is noted. (*B*) Lateral roentgenogram of the skull obtained during the second admission (4 months later) demonstrating, in addition to the previously seen sclerosis of the base, a punched-out 0.5 defect in the left posterior parietal area. This healed spontaneously. (*C*) Magnified view showing sclerosis of occipital bone.

DISCUSSION

"Albright's syndrome" was first reported in 1937 as, "a syndrome characterized by 'osteitis fibrosa' disseminata, areas of pigmentation, and endocrine dysfunction with precocious puberty in females."[1]

In 1942, Lichtenstein and Jaffe[12] added 15 cases of fibrous dysplasia to the 75 they were able to collect from the literature. They described comprehensively the clinical, pathologic and roentgenographic findings.

The clinical manifestations of fibrous dysplasia vary with the extent and location of the skeletal involvement and the presence or absence of extraskeletal manifestations. Albright's syndrome, the most severe form, is usually diagnosed in infancy or childhood. It consists of sexual precocity in the female, cutaneous pigmentations,

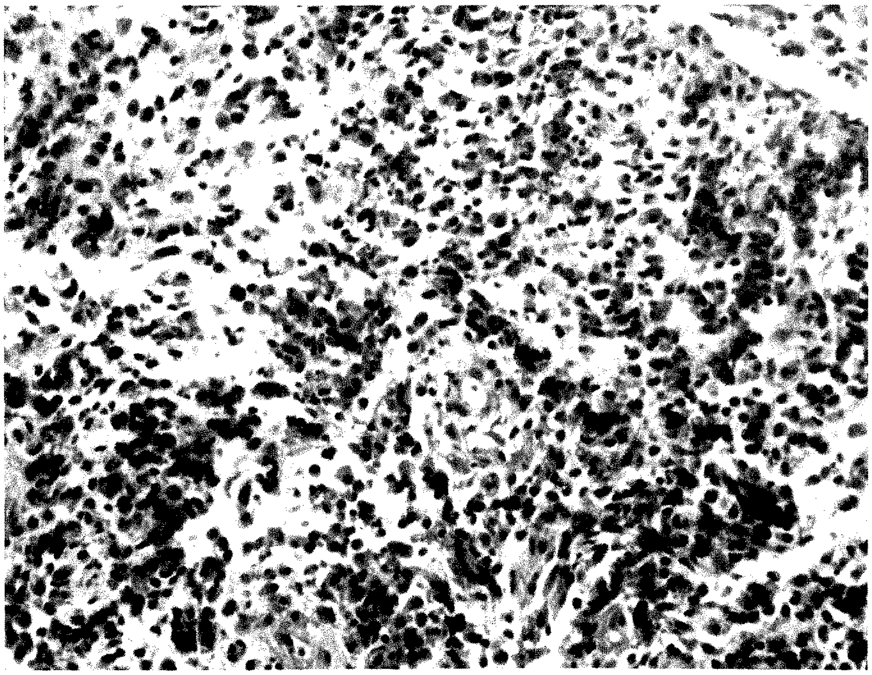

FIG. 5. Photomicrograph of the surgical specimen from the rib showing the characteristic changes of eosinophilic granuloma. The section contains numerous eosinophils and mononuclear histiocytes. Lipid positive macrophages were not present. (H & E, 250×.)

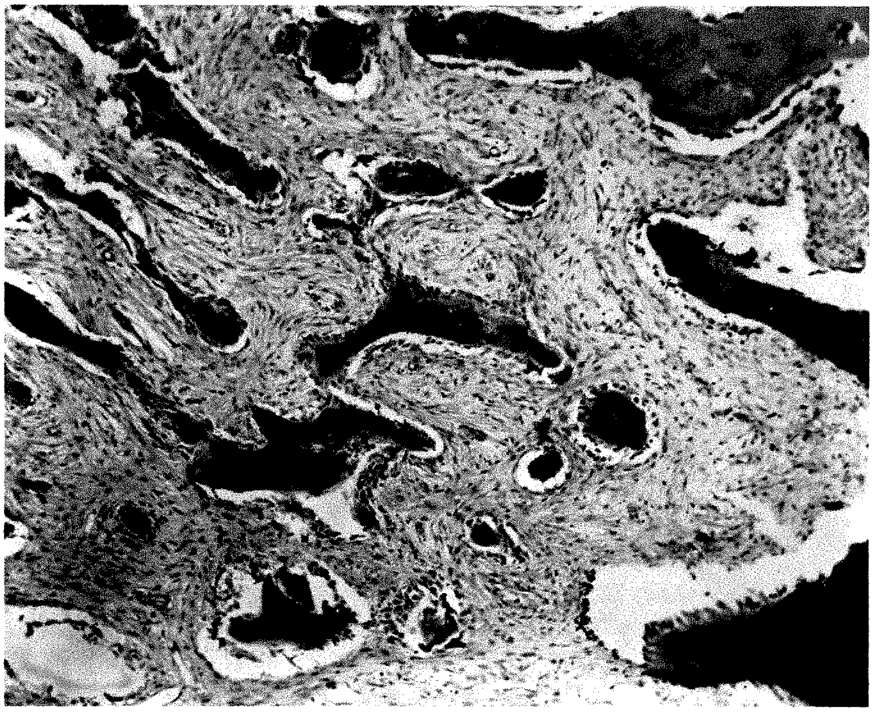

FIG. 6. The pattern of fibrous dysplasia seen in the biopsy of the right iliac crest. The predominant findings were those of whorled fibrous tissue separating and surrounding thin osseous trabeculae. (H & E, 85×.)

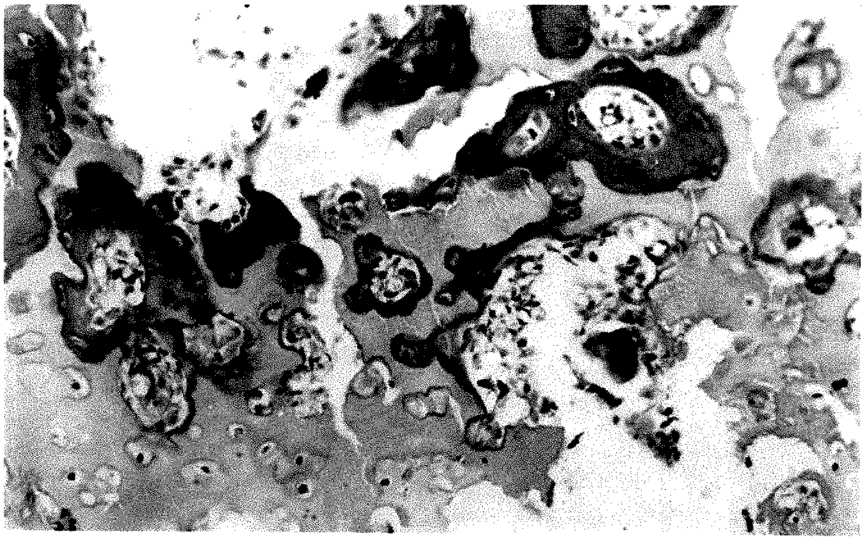

Fig. 7. One of the foci of cartilage present in the specimen illustrated in Figure 6. These "islands" resulted from the irregularities in the linear growth plate observed in the specimen. (H & E, 170×.)

and polyostotic bone lesions which are often unilateral and usually progressive.

The diagnostic histologic component of fibrous dysplasia is whorled connective tissue occupying the marrow space and separating abnormal osseous and, occasionally, cartilaginous elements. The metaplastic bone trabeculae in these areas are thin and disoriented in their relation to normal bone architecture. Considerable morphologic variation may be present with respect to the cellularity of the connective tissue, the degree of vascularization and cyst formation, and the presence of myxomatous or cartilaginous elements. Multinucleated giant cells and foam cells are also variable findings. The proliferating connective tissue replaces the marrow and erodes the cortex. Reactive periosteal new-bone formation does not occur in the absence of a fracture.

The presence of nests of cartilage in the lesions of fibrous dysplasia has long been recognized[1,4,16,17] and has been interpreted as a manifestation of the multipotentiality of fibrous tissue.[12,15] Extensive amounts of cartilage have been found only in cases of fibrous dysplasia associated with precocious puberty.[9] The double pattern of fibrous and "chondrous dysplasia" in Albright's syndrome has been used by Johnson to distinguish this type of fibrous dysplasia from that described by Jaffe and Lichtenstein.[9,10] He feels that the latter form shows little progression after the time of original diagnosis, whether encountered in the young adult or older individual, whereas the form associated with precocious puberty may eventually involve the entire skeleton.

The roentgenographic appearance of the lesions is characteristic and easily recognizable in the polyostotic form of the disease. The lesion replaces the medullary canal and attenuates the cortex from within. Its appearance depends on the degree of ossification and the distribution of the bone trabeculae. If dense and closely knit, an area of sclerosis will result; if rare and scattered, a cyst-like radiolucency will ensue. The typical ground-glass opalescent or smudgy appearance results from the alternation of areas of fibrous tissue with closely meshed trabeculae. Alternating areas of radiolucency and increased opacity are often seen. Thinning of the cortex and consequent expansion of the shaft will occur as the proliferating connective tissue progressively remodels the cortex. Periosteal reaction is not seen without an antecedent fracture. Although the lesions are disseminated, they are focal, and involved bone between the foci of disease retains its normal architecture. A sharp demarcation often exists between the involved and uninvolved areas.

The roentgenographic changes in the skull consist of summation of opalescent or sclerotic lesions, resulting in a marked increase in density of the base of the skull as well as overgrowth and sclerosis of the maxillary bones, the frontal bones and, most typically, marked thickening of the outer table of the occipital bone.

Other roentgenographic features encountered in Albright's syndrome are accelerated bone growth and maturation.

"Eosinophilic granuloma" of bone was described by Lichtenstein and Jaffe[8,11] as single or multiple skeletal lesions and constitutes the least severe form of histiocytosis X. The term, histiocytosis X, is used to describe the reticulo-endothelial alterations present in eosinophilic granuloma, Schüller-Christian disease, and Letterer-Siwe disease. Although occasionally progressive, the lesions of eosinophilic granuloma are more often limited. The variation in pathologic findings of histiocytosis X has recently been reviewed by Lichtenstein.[14] Neither his review nor previous reported series describe an association with fibrous dysplasia.[2,3,8] Conversely, this association has also not been encountered in review of large series of fibrous dysplasia.[15,16,17]

The roentgenographic appearance of histiocytosis X is characterized by sharply marginated radiolucencies, usually showing no calcification within the defects. Although cortical breakthrough with periosteal reaction may occur, expansion of the involved bone is unusual.[2,14] Multiple large or small defects may occur in the calvarium and remaining bones. The lesions of the skull are often preceded by overlying fluctuant subcutaneous nodules.

Growth and bone maturation are usually retarded,[2] and sexual disturbances such as delayed puberty, hypogenitalism, and amenorrhea have been observed.[2,3] This is in sharp contrast to the endocrine disturbances seen in fibrous dysplasia of Albright.

The histologic pattern is also distinctive and consists primarily of histiocytic mononuclear cells and macrophages. A variable number of eosinophils is present in the lesions of eosinophilic granuloma. In Schüller-Christian's disease the histiocytes contain conspicuous amounts of cholesterol and other lipids. Collagen and a granulomatous arrangement of the infiltrate are seen in severe or long-standing cases. Fibrosis was first noted by Green and Farber[7] who interpreted it as a component of healing. Engelbreth-Holm *et al.*[6] postulated morphologic "stages" of histiocytosis X, based on the histologic variation in patients with progressive clinical and roentgenographic involvement.

One controversial aspect of the pathogenesis of histiocytosis X, which is particularly pertinent to the present case, is the mode of resolution of the proliferative lesion of eosinophilic granuloma. Jaffe and Lichtenstein[8,18] have consistently emphasized the lack of fibrosis in the healing of eosinophilic granuloma of bone in the absence of a fracture. They documented a biopsy of an untreated lesion which resolved without fibrosis. Lesions of 2 additional patients have been biopsied with similar findings; these patients, however, had been treated with steroids.[5] It appears that when intracellular lipid is present in lesions of histiocytosis X, collagen may accompany the evolution of the infiltrate.[7,18] Proliferative lesions which contain no lipid may resolve without further progression and without fibrosis as part of their repair.

The remote possibility of widespread unilateral histiocytosis X in the patient presented, resolving with a roentgenographic and histologic pattern simulating fibrous dysplasia, is entirely unlikely. The characteristic roentgenographic findings of fibrous dysplasia, the presence of suggestive columns of cartilage, and the clinical manifestation of Albright's syndrome all point to the contrary. The typical punched-out defect in the parietal bone which healed spontaneously and the roentgenographic and histologic appearance of the rib lesion similarly indicate that these 2 lesions were those of eosinophilic granuloma. Moreover, radiologists who have occasion to observe long-term follow-up of healing or healed bone lesions in histiocytosis X are aware that the altered architecture seen at the site of previous lesions does not simulate fibrous dysplasia. Lesions of eosinophilic granuloma can, in fact, heal completely without any residual roentgenographic changes.

Although both conditions may be protean, we believe that the patient presented here exemplifies an unusual association of concomitant diseases, rather than demonstrating a relationship between the two entities.

SUMMARY

A female patient with a fortuitous association of Albright's syndrome and eosinophilic granuloma is reported.

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◆

A SIMPLE SCREENING TEST FOR THE MARFAN SYNDROME*

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THERE are so many lanky, thin persons who have spidery fingers, wear thick-lensed glasses, and appear prematurely old—characteristic features of the Marfan syndrome—that a simple, quick screening test to truly distinguish them from those with the Marfan syndrome is of significant importance. Protrusion of the thumbs beyond the confines of the clenched fist (Fig. 1*A*) in a patient with the Marfan syndrome was first reported by Parker and Hare[2] in 1945. For many years, this test has been used in this center and has been found to be reliable in patients with classic skeletal, ocular, and cardiovascular signs of the Marfan syndrome (Fig. 2, *A-H*).

In only 3 of several hundred patients examined was protrusion of the thumbs beyond the clenched fist present without other (cardiovascular and ocular) confirmatory signs of the Marfan syndrome. In 2 of these, roentgenography of the clenched fists disclosed that the tip of the long digits of the thumb was within the confines of the bony skeleton of the hands, whereas it protruded beyond this point in the patients with the Marfan syndrome. In this report the sign is described, the falsely positive cases are reported, and a recommendation is made that elicitation of this sign should become a part of the routine physical examination.

METHODS AND MATERIALS

Over 10 patients with proved Marfan's syndrome[3-7] had a positive thumb sign (Fig. 1*A*). This was elicited by having the patient make a fist over the thumb. When positive, the thumb clearly extended beyond the confines of the fist (Fig. 1*A*). Forceful and undue stretching of the thumb in order to make it protrude is an undesirable maneuver, but rarely produces a falsely positive test. Of the patients examined at

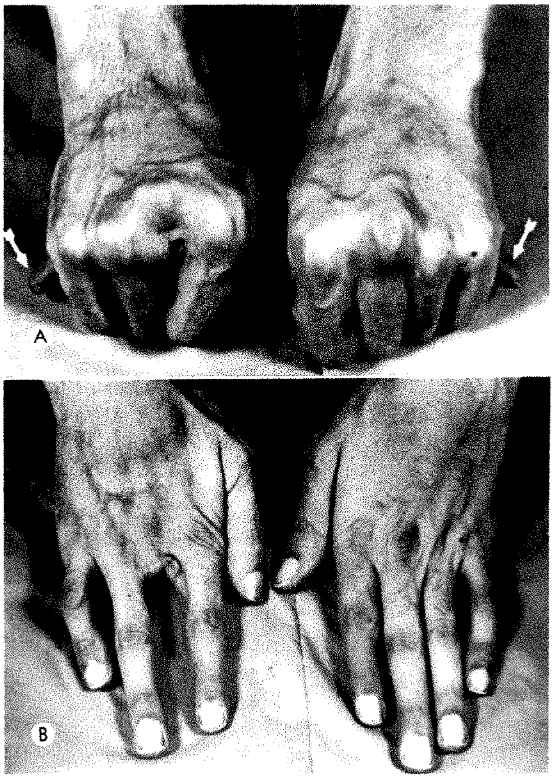

FIG. 1. *Autopsy proved case of the Marfan syndrome in a 21 year old man who died of dissecting aneurysm of the ascending aorta following insertion of a prosthetic mitral valve resected because of severe mitral insufficiency following bacterial endocarditis.* (*A*) Protruding thumbs (arrows) beyond the confines of the clenched fists. (*B*) Photograph of fingers (arachnodactyly). The right middle finger had been amputated following trauma but this deformity did not prevent the positive test. Courtesy of Cohen and Kaye.[1]

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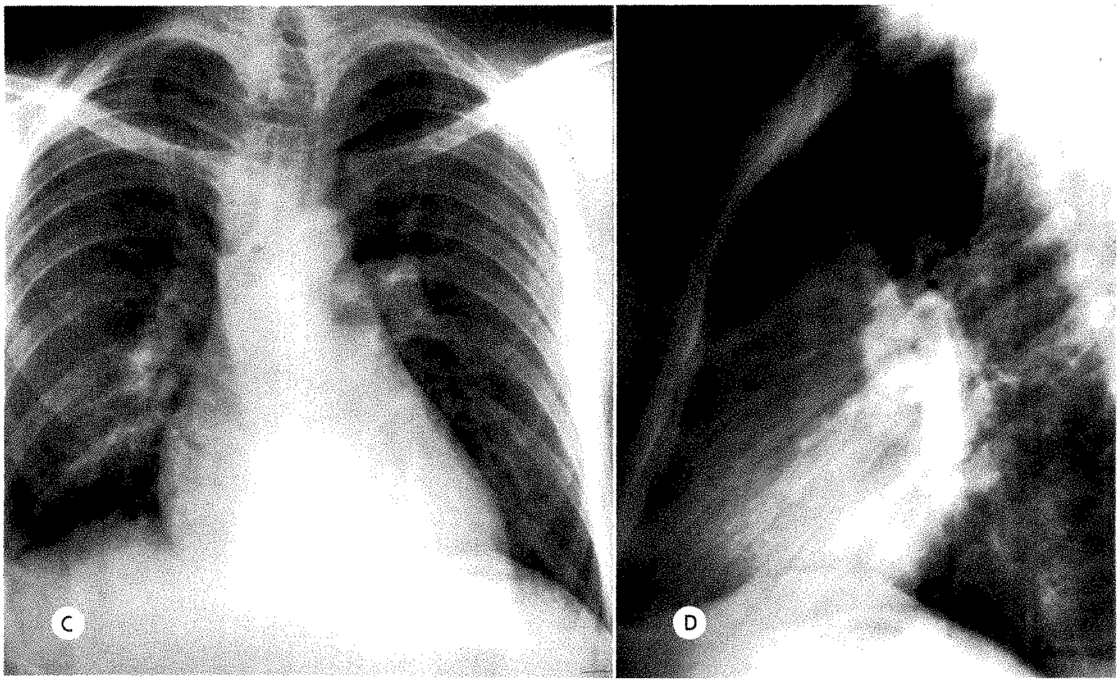

FIG. 1. (*C*) Frontal teleroentgenogram of the chest showing spinal scoliosis and cardiac enlargement. (*D*) Lateral view showing the long thorax.

this center for this sign, both in the clinic and in the hospital, only 3 patients appeared to have a falsely positive sign. One patient had the skeletal changes of the Marfan syndrome (Fig. 3, *A–E*), but aortic stenosis rather than regurgitation was present. Insertion of the aortic valve prosthesis alleviated severe heart failure (paroxysmal nocturnal dyspnea). In this case, the bony tip of the thumb cleared the outer margins of the bony fist (Fig. 3*E*). In 2 other patients with a false positive sign, roentgen examination of the clenched fist showed that the osseous portion of the thumb was within the confines of the fist and did not extend beyond the bony tissues of the hand (Fig. 4*C* and 5). In the first case, a woman of 20 years, there were no skeletal, eye, or cardiovascular malformations but she had been an acrobatic dancer for many years, was "double jointed," and exercised by wrapping her knees about her head. The second of these 2 patients had a high arched palate and myopia without scoliosis of the spine or a cardiac murmur.

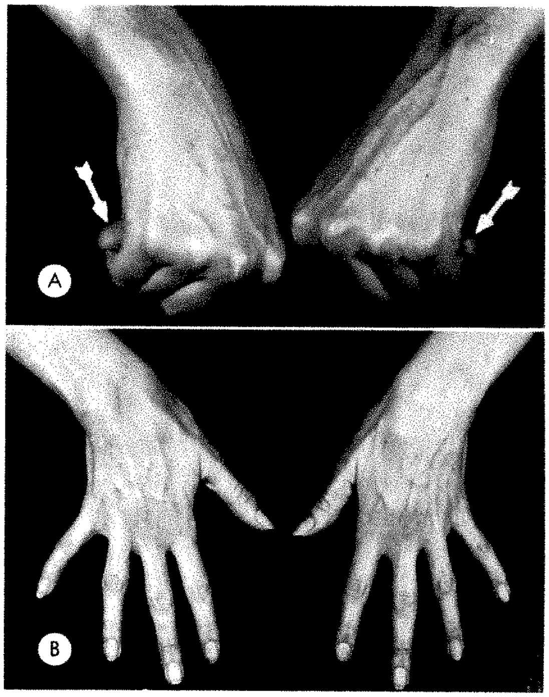

FIG. 2. *Classic Marfan syndrome in a 26 year old woman.* (*A*) The thumb sign is positive (arrows). (*B*) Photograph of hands showing arachnodactyly.

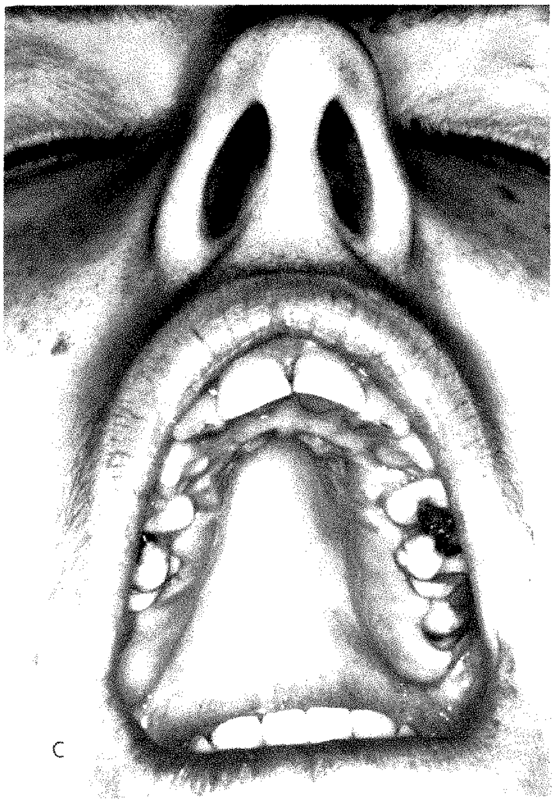

Fig. 2. (*C*) Photograph showing the high arched palate.

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DISCUSSION

In well established proved cases of Marfan's syndrome with its classic skeletal, ocular, and cardiovascular manifestations, protrusion of the thumbs beyond the clenched fist was evident (Fig. 1*A* and 2*A*). Two patients with dissecting aneurysms, but with none of the skeletal or ocular abnormalities associated with Marfan's syndrome, had negative thumb signs. In 1 of these patients, medial cystic degeneration was noted at surgery.[7] In the patient with a positive thumb sign (Fig. 3*A*) and calcific stenosis, only the calcific portion of the aortic valve was excised, and this did not contain sufficient aortic wall to determine if medial cystic degeneration was present. The roentgenogram of her clenched

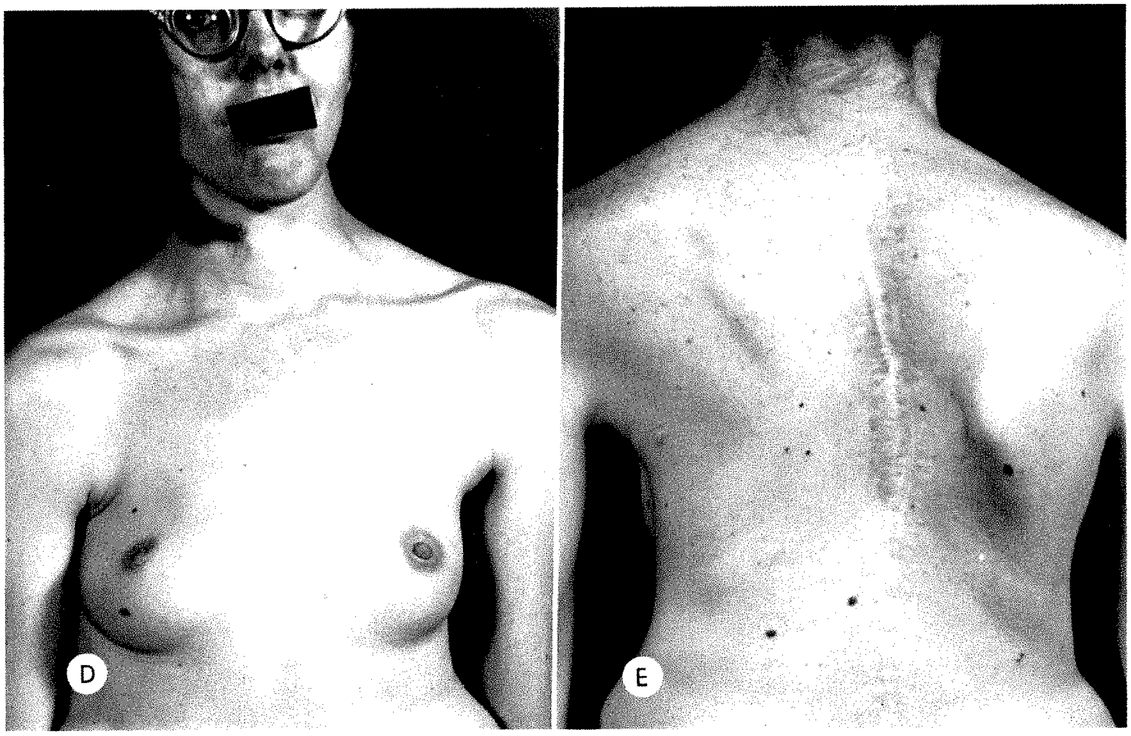
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Fig. 2. (*D*) Frontal photograph showing the thick lenses (worn because of dislocated lenses and myopia) and the deformed thorax. (*E*) Dorsal photograph showing the spinal deformity and scars of previous operations for correction of scoliosis.

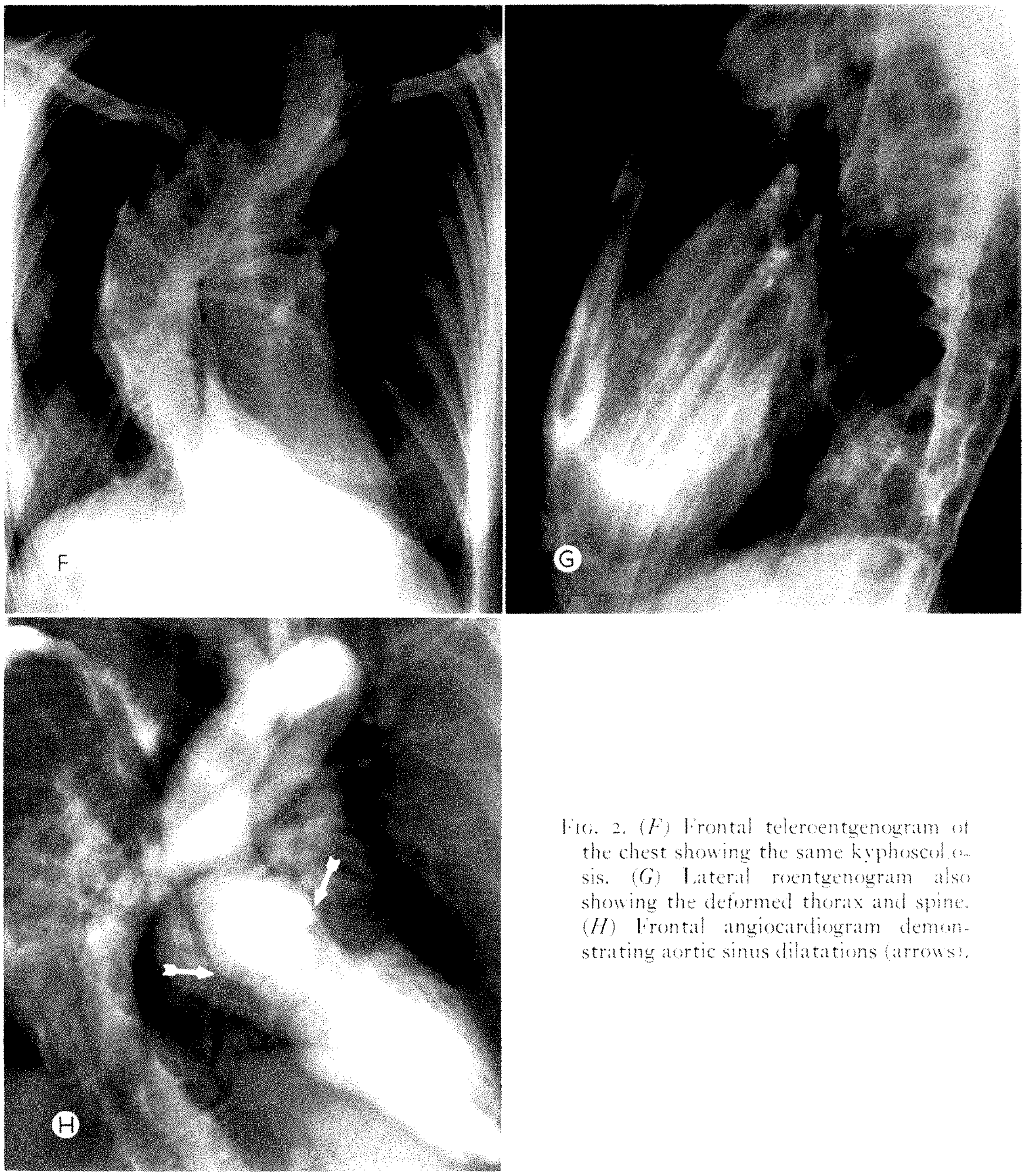

Fig. 2. (*F*) Frontal teleroentgenogram of the chest showing the same kyphoscoliosis. (*G*) Lateral roentgenogram also showing the deformed thorax and spine. (*H*) Frontal angiocardiogram demonstrating aortic sinus dilatations (arrows).

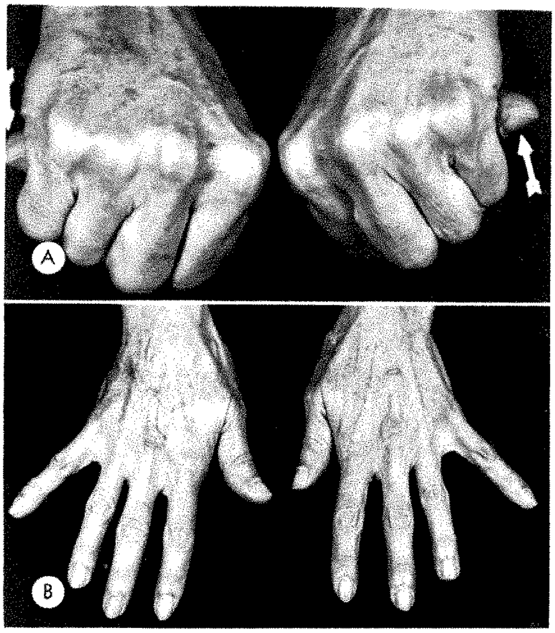

Fig. 3. *Positive thumb sign in a 57 year old woman with arachnodactyly, scoliosis of the spine, and myopia with calcific aortic stenosis corrected by prosthesis.* (*A*) Photograph of positive thumb sign (arrows). (*B*) Photograph of fingers showing long slender digits.

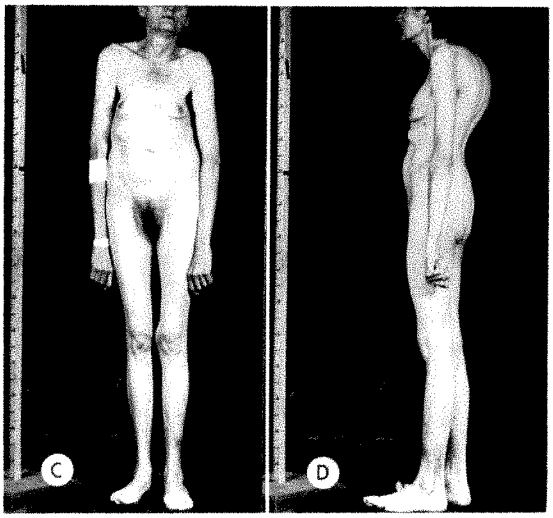

Fig. 3. (*C*) Frontal photograph showing the long lanky build with long arms and legs. (*D*) Lateral view shows the kyphoscoliotic deformity of the thoracic spine.

fists showed that the thumbs protruded beyond the bony fists (Fig. 3*E*). In 2 other patients, the thumb sign appeared to be positive, but the roentgen study of the clenched fists revealed that the phalangeal tips of the thumbs were within the fleshy fist (Fig. 4*C* and 5). Since angiocardiography was not performed in these patients, cardiovascular disease, especially dilatation of the aortic sinuses, cannot be ruled out with certainty.[3–7] The history of double-jointedness from acrobatic dance training would probably account for the unusual mobility of the thumbs within the clenched fists in 1 of the patients. The other patient, although he had a huge arched palate, was without spinal deformity or cardiac murmurs. It is anticipated that patients with Ehlers-Danlos syndrome may have positive

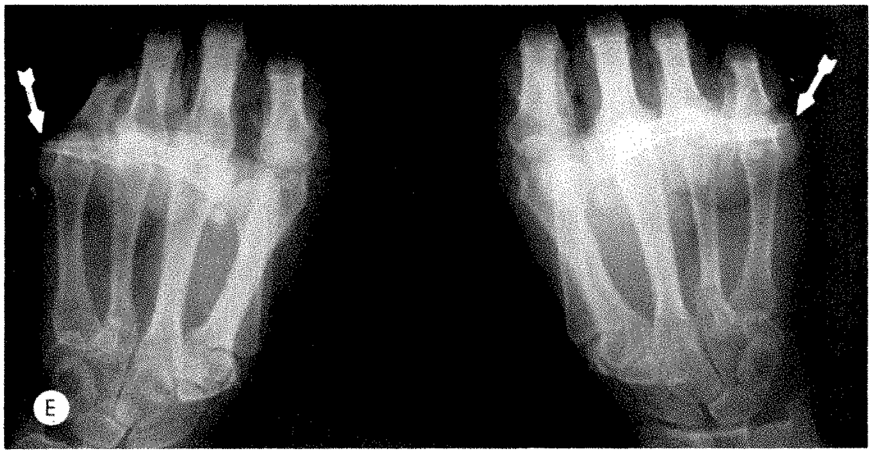

Fig. 3. (*E*) Roentgenogram of the clinched fists shows that the terminal digits of the thumbs protrude beyond the confines of the clenched fists (arrows).

thumb signs because they also have hypermobility of their joints; however, none has been studied as yet.

SUMMARY AND CONCLUSIONS

A protruding thumb beyond the confines of the clenched fists appears to be a confirmatory sign of the Marfan syndrome. In all of the 10 patients with the classic skeletal, ocular, and cardiovascular features of Marfan's syndrome, the test proved positive. Only 3 false positive tests were encountered so far in a large number of patients during routine physical examinations. In 1 patient with a markedly positive test, the skeletal features of Marfan's syndrome were evident but, instead of aortic regurgitation, aortic stenosis was found, and was alleviated by insertion of a prosthetic valve. In 2 other patients with mildly protruding thumbs, roentgen examination of the clenched fists revealed that only the soft tissue (fleshy portion) of the thumb protruded beyond the bony portions of the fists, while the bony tip of the terminal thumb phalanx was within the confines of the metacarpal bones of the fists. Two patients with dissecting aneurysms but without other manifestations of the Marfan syndrome had negative thumb signs.

It is, therefore, proposed that the clenched fist test to see whether protrusion of the thumb is present be made a routine part of the physical examination. When positive, confirmatory findings, such as double-jointedness, arachnodactyly, ster-

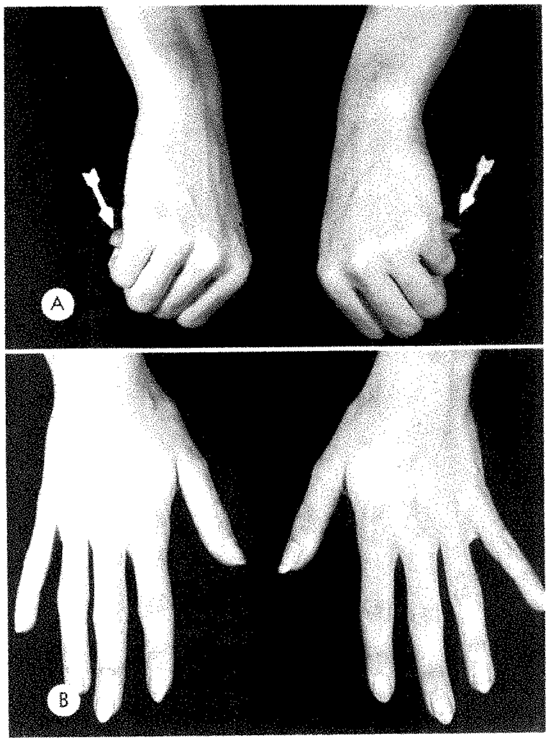

Fig. 4. *False positive thumb sign in a 20 year old acrobatic dancer without skeletal, eye, or cardiac manifestations of the Marfan syndrome.* (*A*) Photograph of clenched fists showing mild protrusion of thumbs (arrows) beyond the clenched fists. (*B*) The fingers of the hands appear long and narrow.

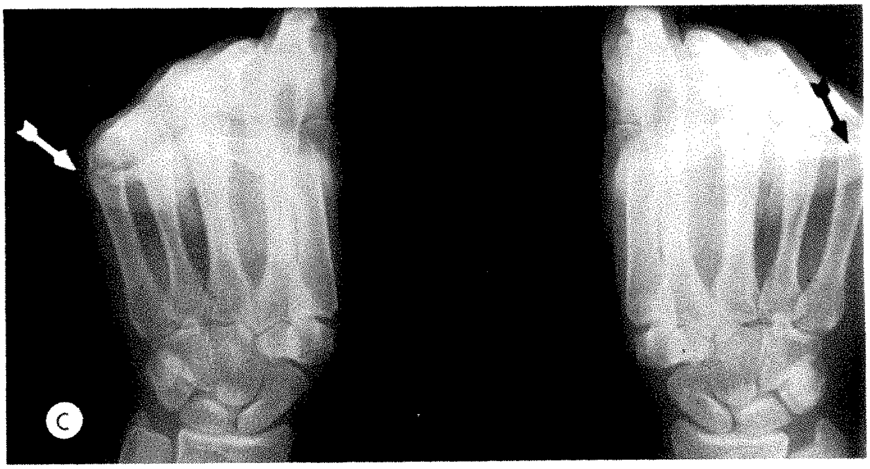

Fig. 4. (*C*) Roentgenogram of the clenched fists, however, shows that the terminal digits of the thumbs (arrows) lie well within the fist and indeed do not extend beyond the outer border of the metacarpal bones.

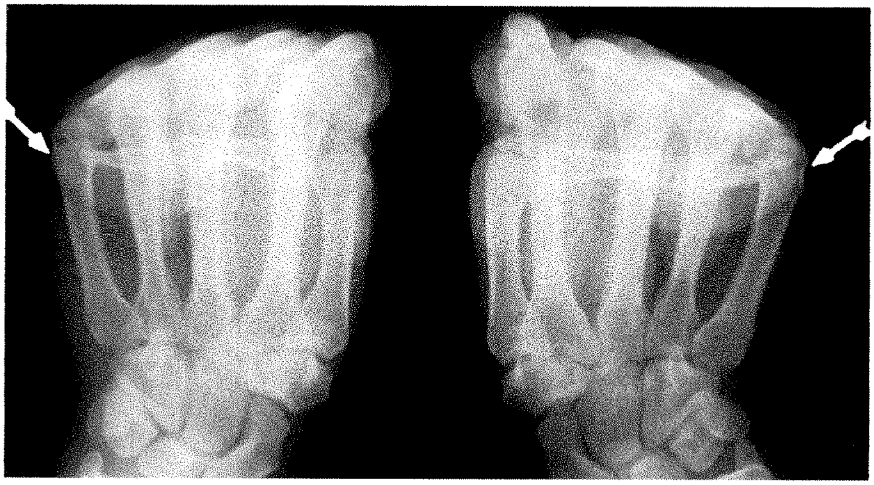

FIG. 5. *False positive thumb sign in a 27 year old man with myopia and high arched palate but without other skeletal or cardiac manifestations of the Marfan syndrome.* Roentgenogram of the clenched fist discloses that the bony digits of the thumbs (arrows) are well within the clenched fists.

nal, spinal, and skull (dolichocephaly) abnormalities, a high arched palate, ocular disease (ectopia lentis, myopia, and colobomas) and cardiovascular (aortic sinus dilatation, aortic insufficiency, aortic and pulmonary arterial aneurysms, and aortic dissection) should be sought for. Rarely, all these findings may be absent and only the thumb sign may be positive. In such instances, roentgen examination of the clenched fists is advised because double-jointedness in acrobatic persons apparently produces unusual mobility without other skeletal abnormalities. In the patient discussed, the terminal phalanx of the protruding thumbs, in contrast to that in patients with the Marfan syndrome, did not extend beyond the confines of the metacarpal bones of the clenched fist.

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TEMPORAL BONE ABNORMALITIES IN CONGENITAL NEUROSENSORY DEAFNESS AS REVEALED BY PLESIOSECTIONAL TOMOGRAPHY*

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THE etiology of congenital deafness has always been of considerable interest to those physicians and workers in allied specialties who deal with this problem. Uncertainty as to the site of the abnormality has led to difficulty in treatment and training of patients affected with this condition. The ability to localize the site of abnormality can be of great importance in planning the type of treatment or training which can provide the best results in aural habilitation.

Prior to the 1950's the locus of otologic pathology was assessed primarily by otologic and audiologic examinations and, to a lesser extent, by roentgenologic techniques. The usual roentgen examination of the skull and mastoid region was the source of most anatomic information about the otic capsule region. In children, particularly the younger age group, evaluation of the temporal bone area by routine roentgenography is often limited because of the necessary intricate positioning and angulation of both the roentgenographic tube and the patient. Examination is frequently limited to a modified lateral view of the mastoids, such as the Runström or Law view, a Chamberlain-Towne projection and a submentovertex view. Older children and adults can be examined more thoroughly. The examination in this group may consist of a full skull and mastoid study. The routine skull study in our department includes stereoscopic examination in the posteroanterior position, the Chamberlain-Towne position, and the lateral projection. Our usual mastoid study consists of single roentgenograms in the submentovertex position, Chamberlain-Towne views (omitted if the skull is examined at the same time), the Mayer view and the Stenvers views of both temporal bones. In addition, a stereoscopic Runström view is obtained. Several authors have felt that the routine views of the temporal bone were adequate and satisfactory for thorough evaluation of the otic capsule area.[22]

These views do provide a moderate amount of information about the region of the middle and inner ear, but the region of the oval window, ossicular mass and cochlea is ordinarily not well seen. We feel that with the development of more refined roentgenographic procedures, such as polytomography and multisection tomography, the fine detail of the temporal bone has been more easily visualized and abnormalties identified with more certainty.[8,6,11,14,16,19,21,24,25]

With the development of plesiosectional tomography by McGann[19] in 1961, a simple method became available for excellent roentgenographic demonstration of the component portions of the temporal bone. This technique, a refinement of linear multisection tomography, provides 4 sections approximately 1 mm. apart for each roentgenographic exposure. Ordinarily, 2 roentgenographic exposures are made for each temporal bone. The patient is placed supine on the roentgenographic table and the head immobilized so that Reid's base line is in a vertical position. The central beam is centered to the lateral canthus of the eye. The initial exposure is obtained with the tomographic fulcrum at the root of the tragus of the external ear. A second exposure is centered 0.5 cm. posterior to this point.

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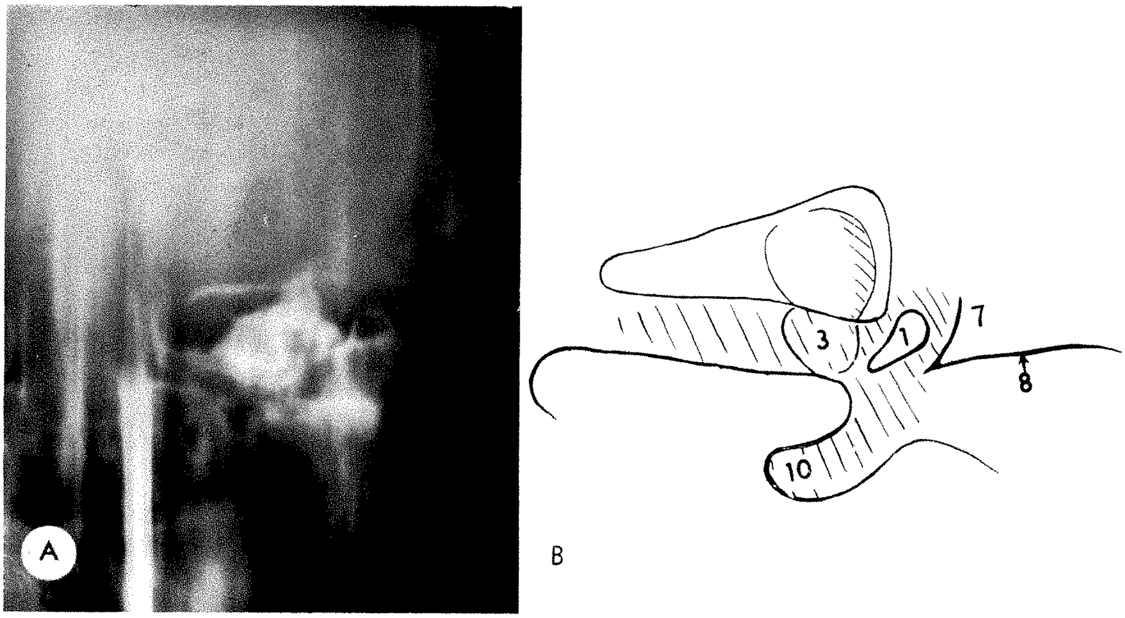

Fig. 1. (*A* and *B*) Median cut gives good detail of ossicles (1), vestibule (3), attic wall (7), and roof of external auditory canal (8), as well as the oval window. The medially extending hypotympanum (10) is seen beneath the promontory of the cochlea.

These two exposures provide 8 sections through the temporal bone and almost always reveal the middle and inner ear structures satisfactorily.

The cassette needed for plesiosectional tomography is a modification of the usual roentgenographic cassette and contains 4 sets of matched intensifying screens. These are now available commercially.

It has been reported that approximately 65 per cent of American radiologists have a linear tomographic device available for their use,[17] thus making plesiosectional tomography applicable for use by this group.

The immediate advantage of plesiosectional tomography over polytomography is that ordinarily only 2 roentgenographic exposures are made on each side as opposed to 10 to 15 by the Polytome method reported by Everberg.[7,8] Other users of the Polytome take 15 to 18 exposures of both sides simultaneously. The reduction in roentgenographic exposure using plesiosectional tomography is obvious. In addition, the plesiosectional tomography exposure is of 2 seconds duration as opposed to 6 seconds on the Polytome. This has meant that no anesthesia and little or no sedation have been needed in almost all the children in this group examined by plesiosectional tomography. These are frequently necessary when using a long exposure and immobilization time. In polytomography, the procedure is prolonged because of the necessity to await roentgenographic tube cooling after each 6 exposures done at a rate of 2 per minute.

Since 1952, several radiologists have reported on the normal and abnormal findings in the middle and inner ear regions. Samuel and Theron[22] described the roentgenographic appearance of the ossicular structures and the findings in several abnormal cases, but felt that tomography was of no value. Frey and Mündnich[11] in 1957 described in detail some of the abnormal bone findings in deafness and other otologic problems, and Everberg *et al.*[7,8,9,10] have published several articles on their investigations in cases of unilateral deafness in children; these investigators utilized tomography.

Other abnormal findings in the temporal

bone of patients with deafness have also been reported by Camp and Allen,[5] James *et al.*[13] and Soila.[23]

With the availability of plesiosectional tomography, it was decided to study roentgenographically the temporal bones of a large group of children with congenital neurosensory deafness. These children were being seen by the otologic and audiologic service of St. Christopher's Hospital for Children, Philadelphia, Pennsylvania.

Table I

KEY TO FIGURES 1 THROUGH 5

1. Ossicles
2. Oval window
3. Vestibule
4. Semicircular canals
5. Internal auditory canal
6. Crista falciformis
7. Attic wall
8. Roof of external auditory canal
9. Promontory
10. Hypotympanum
11. Cochlea

This facility provides a center for the evaluation of auditory problems, recommendation of therapy for the condition involved and at least a start toward aural habilitation or rehabilitation, depending upon the etiology of the underlying abnormality. All of the patients seen are children, although some of their parents with similar conditions have also been evaluated by us in an attempt to discover any possible hereditary or familial link. A large percentage of this group of children has neurosensory hearing loss. The diagnostic facilities include complete pediatric, otologic and audiologic evaluation. The evaluation of the type of hearing loss present leads to recommendations concerning the type of hearing aid to be used and the best type of training program for the individual involved. If hearing loss is found to be irreversible, a language training program is instituted. This involves a multisensory approach to communication involving kinesthetic sense, visual, tactual and, if possible, any auditory sensation present. In cases with bone abnormalities of the temporal area associated with hearing loss, the use of hearing aids has been eliminated since these patients are found not to respond.

ROENTGEN FINDINGS

Everberg[7,8] has reported several groups of roentgenographic abnormalities of the temporal bone. These included the absence of the inner ear cavity or portions thereof, absence of the cochlea with the vestibule and lateral semicircular canal making up one large cavity and atresia of the internal

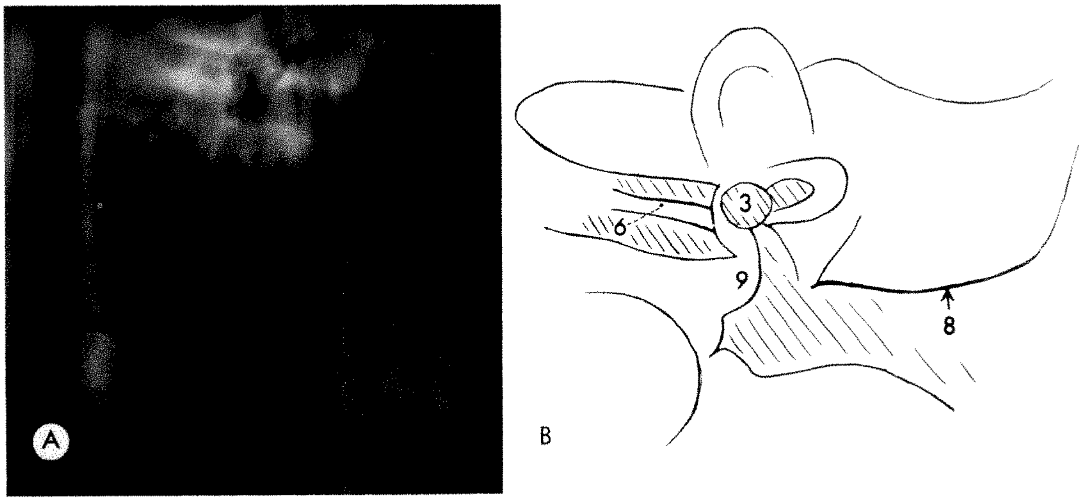

Fig. 2. (*A* and *B*) A more posterior section shows the crista falciformis (6) to good advantage in the medial portion of the internal auditory canal. The vestibule (3) and promontory (9) are also well seen.

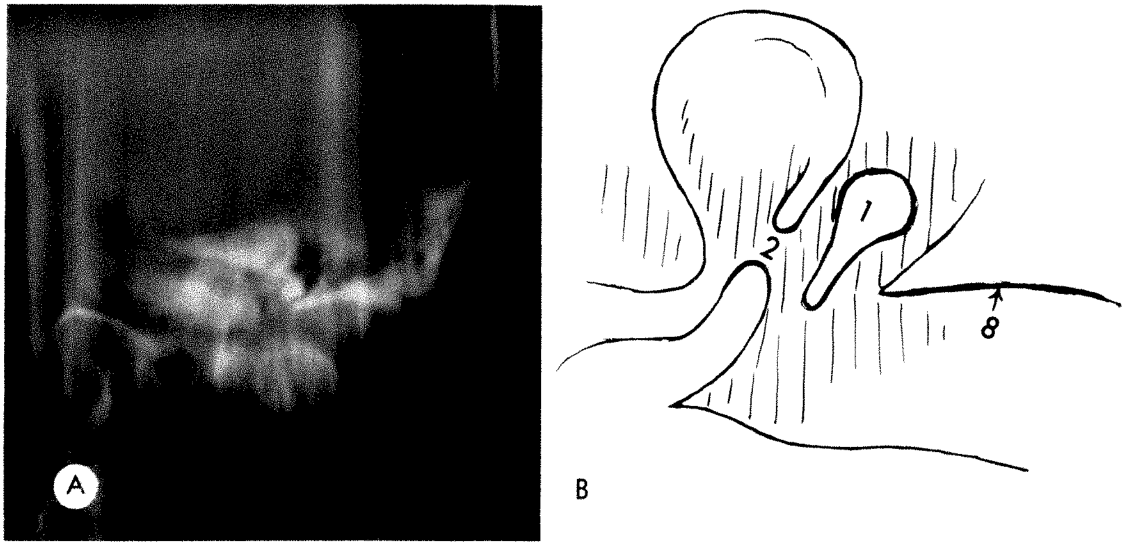

FIG. 3. (*A* and *B*) The vestibule, semicircular canals and oval window (2) are satisfactorily visualized as are the ossicles (1) and roof of the external auditory canal (8).

auditory canal.[8] Many of his cases of unilateral deafness were felt to be of congenital origin. Others were thought to represent residua of various forms of infection.

Knowledge of the normal anatomic structures in the temporal bone is vital to an understanding of the abnormalities which may be encountered.

Plesiosectional tomography of the temporal bone reveals considerable anatomic detail in the normal patient.

The bony margins of the external auditory meatus and canal can be well visualized. The bony canal is the medial two-thirds of that structure and in the anteroposterior projection this bony tube is slightly irregular and narrows medially.

The superior portion of the tympanic membrane is attached to the medial extent of the roof of the external canal (Fig. 1, *A* and *B;* 2, *A* and *B;* and 3, *A* and *B*; Table 1, no. 8). From this point, a short bony plate can be seen extending superolaterally. This represents the lateral wall of the attic. (Fig. 1, *A* and *B;* Table 1, no. 7).

Occasionally, the tympanic membrane itself can be identified, since it contrasts with air on either side.

The middle ear or tympanic cavity presents as a relatively radiolucent area since it is air-containing and slightly deeper in anteroposterior dimension than the adjacent external canal. The ossicular mass can be seen in its upper portion (Fig. 1, *A* and *B;* 3, *A* and *B;* and 4, *A* and *B;* Table 1, no. 1). In the anteroposterior projection a considerable portion of the malleus and incus are superimposed. The head of the malleus obscures the body of the incus and the manubrium lies anterior to the long process of the incus. The long process of the incus is occasionally seen protruding medially toward the oval window. Rarely, the short process of the incus can be seen lateral to the head of the malleus. Although the presence of the stapes can be suspected on occasional plesiosectional tomograms of the temporal bone, we do not feel that this structure is visualized satisfactorily enough to make any statements as to its position or condition.

The hypotympanum and origin of the eustachian tube can be identified on anterior sections through the middle ear cavity. The hypotympanum, appearing beneath the promontory of the cochlea (Fig. 2, *A* and *B;* Table 1, no. 9) on these sections, varies considerably in extent and configuration. This region is occasionally quite large, giving the middle ear cavity a medial extension rather than its usual tilted, but generally rectangular, configuration (Fig. 1,

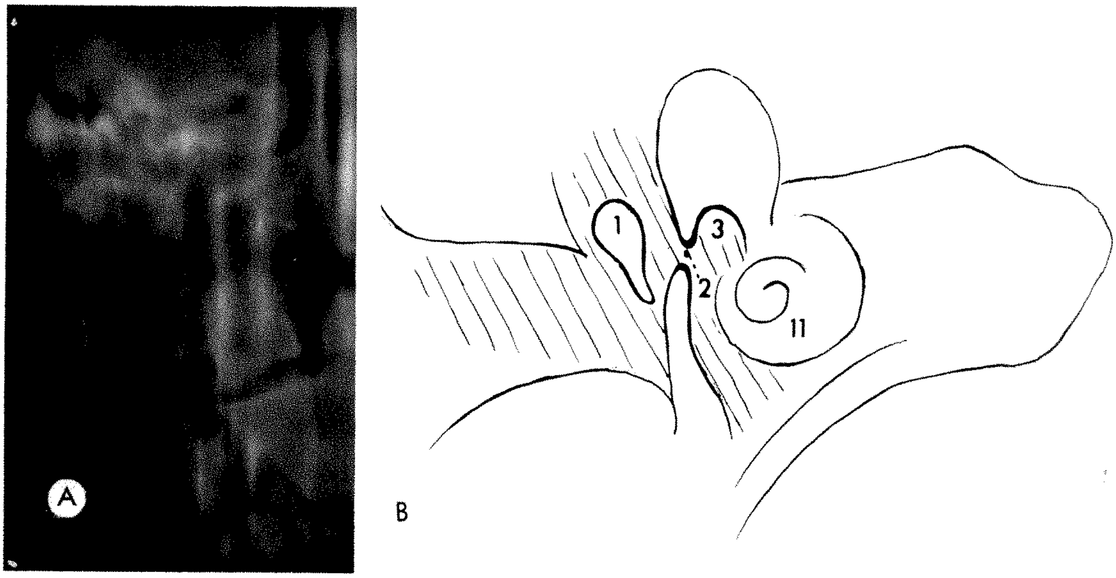

Fig. 4. (*A* and *B*) The cochlea (11) is better seen on this more anterior section. The ossicles (1), oval window (2) and vestibule (3) are also satisfactorily delineated.

A and *B;* and 5, *A* and *B;* Table 1, no. 10).

The jugular fossa also shows a variable appearance. Ordinarily, the region of the jugular bulb lies beneath the posterior portion of the medial aspect of the middle ear cavity and is separated from it by a thin bony plate (Fig. 6, *A* and *B*).

The bony portion of the pharyngotympanic canal or eustachian tube can often be seen extending medially in an almost horizontal course beneath the promontory of the cochlea, in sections anterior to the jugular fossa and posterior to the carotid canal. More anterior sections show the carotid canal as it forms the medial aspect of the anterior wall of the tympanic cavity.

As noted earlier, the ossicles can be seen in the attic region between the tympanic antrum and tympanic cavity. The antrum itself is an irregular radiolucent area usually measuring approximately 8–12 mm. in diameter. This space can be seen in the anteroposterior projection just lateral to and above the level of the lateral semicircu-

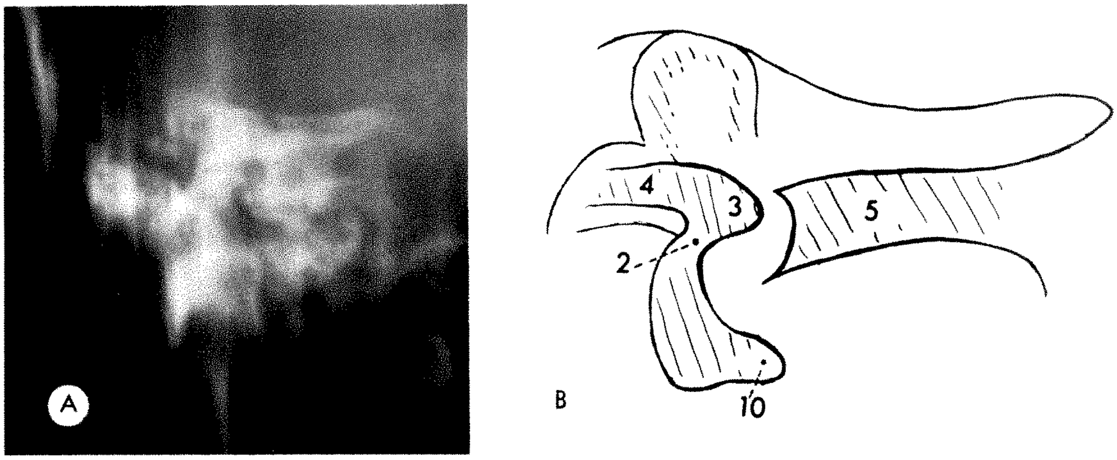

Fig. 5. (*A* and *B*) This more posterior section shows the internal auditory canal (5) with the posterior lip of the meatus well visualized. The promontory with the hypotympanum (10) below the oval window (2), vestibule (3) and semicircular canals (4) above are all well seen.

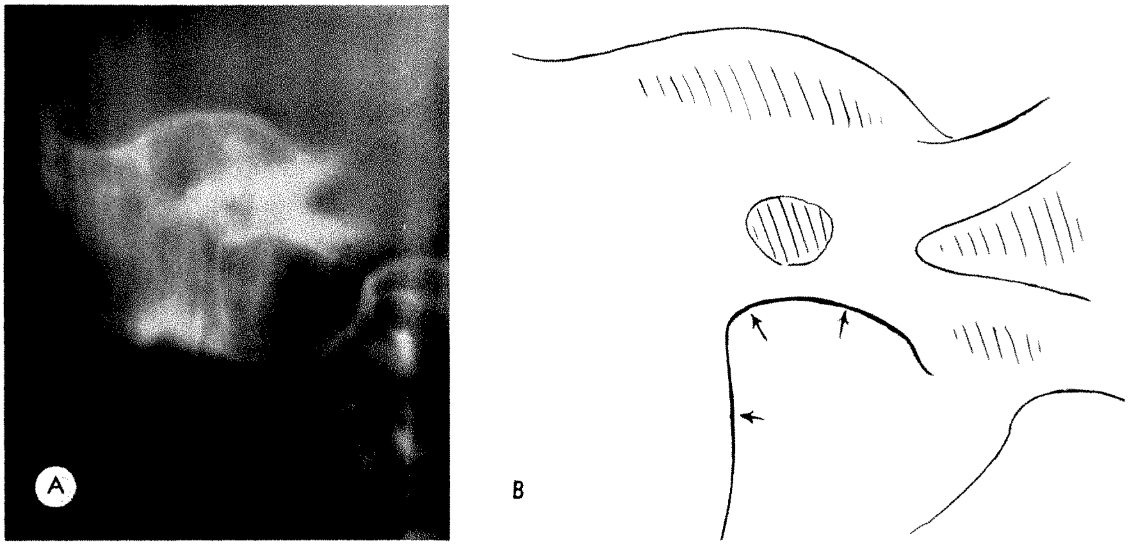

Fig. 6. (*A* and *B*) A further posterior section discloses the superior margin of the jugular fossa (arrows) forming the medial portion of the floor and posterior wall of the middle ear space.

lar canal. Measurements above 12 mm. in diameter in this projection found in patients with sclerotic or diseased mastoid air cells should suggest the possibility of bony breakdown and cholesteatoma formation.[2]

The bony labyrinth lies medial to the tympanic cavity and consists of the semicircular canals, vestibule and cochlea.

Only the lateral and superior semicircular canals are visualized, while the posterior semicircular canal is usually not outlined by this method of study. The canals, as well as the remainder of the bony labyrinth, are visualized as relatively radiolucent structures because of their soft tissue content and bony supporting structure (Fig. 5, *A* and *B;* Table 1, no. 4). At the junction of the superior and lateral semicircular canals lies the bony vestibule which contains the sacculus and utriculus. Roentgenographically, the vestibule is a round radiolucent structure, measuring 2 to 3 mm. in diameter in the normal temporal bone (Fig. 1, *A* and *B*; 2, *A* and *B*; 4, *A* and *B*; and 5, *A* and *B*; Table 1, no. 3). Enlargement of this region has been seen in patients with neurosensory deafness as will be described later. On the lateral aspect of the vestibule a break in the bony ring can be seen in approximately 80 per cent of patients examined by plesiosectional tomography. This apparent break in the bony ring is due to the presence of the oval window or fenestra vestibuli (Fig. 3, *A* and *B*; 4, *A* and *B*; and 5, *A* and *B*; Table 1, no. 2). The

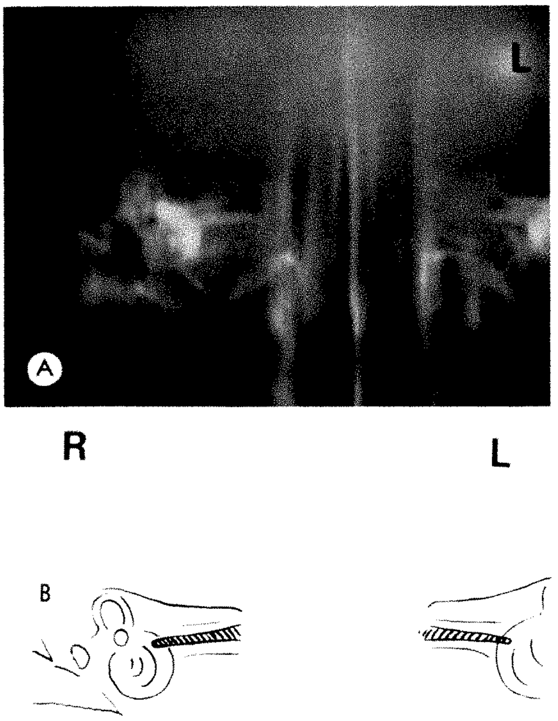

Fig. 7. (*A* and *B*) A child with bilateral congenital neurosensory deafness shows marked narrowing of the internal auditory canals. These structures measure less than 1.5 mm. in height.

discontinuity of the bone is rarely over 1.5 mm. in height. Occasionally, the bony ring will appear continuous but thinned in this region and it may be that the foot plate of the stapes is being visualized since that structure normally occludes the oval window.

The bony cochlea itself can be well visualized on the more anterior sections (Fig. 4, *A* and *B*; Table 1, no. 11). The spiral lamina of this organ can be easily identified by its unmistakable appearance. The medial portion of the cochlea overlies the internal auditory canal in the anteroposterior projection. The coils of the spiral lamina can be followed and variations in its structure have been found to be significant in congenital deafness problems.

The roentgenographic appearance of the internal auditory canal has been thoroughly reviewed by several authors,[4,11,14,21,25] and the components of its structure are well delineated in these examinations (Fig. 5, *A* and *B*; Table 1, no. 5). The lateral bony wall or lamina cribrosa, which is common also to the vestibule, is perforated for the passage of nerve fibers from the otic labyrinth to the internal auditory canal. These

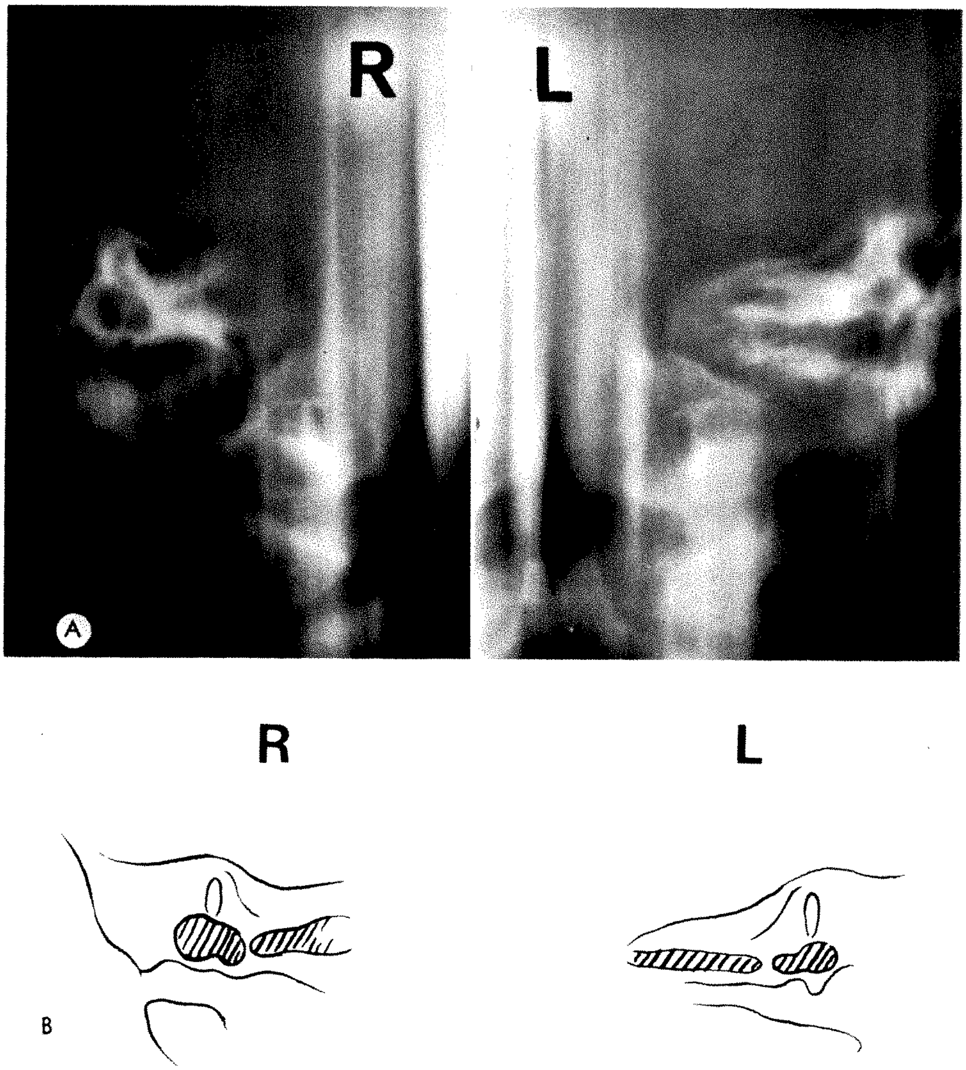

Fig. 8. (*A* and *B*) Bilateral congenital neurosensory deafness with marked enlargement of the right vestibule and incorporation of the horizontal and a portion of the superior semicircular canal within it. The right internal auditory canal is narrow. The left internal auditory canal is quite narrow and presents a slit-like appearance.

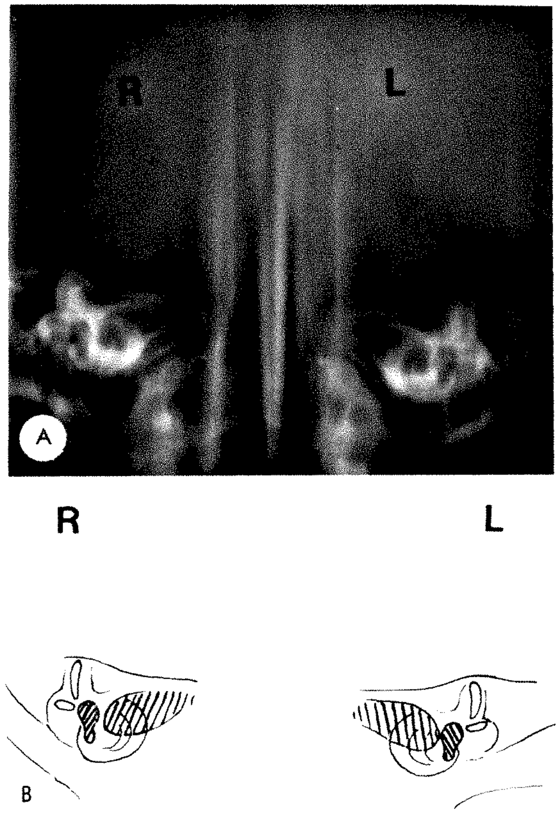

Fig. 9. (*A* and *B*) One of 2 cases showing marked widening and shortening of the internal auditory canals. Both bony vestibular margins are enlarged.

perforations cannot be visualized roentgenographically. The important point of the orientation of the meatus has been thoroughly reported upon by Valvassori and it is necessary for the radiologist to realize that we are only seeing the posterior lip of the meatus, while the anterior edge blends with the petrous apex.

The crista falciformis or bony plate separating the seventh and eighth nerves in the canal is frequently well demonstrated (Fig. 2, *A* and *B;* Table I, no. 6).

The size of the internal auditory canal is felt to average 4 to 5 mm. in height with less than 1 mm. in difference between the sides in almost all patients. The length of the canal measured from the lamina cribrosa or bony plate separating the vestibule from the internal auditory canal averages 7 mm. to the posterior lip with occasional variation of less than 2 mm. between the sides. Variations of more than 2 mm. should be viewed with extreme suspicion since the thinner posterior wall may be the initial site of demonstrable bony destruction by eighth nerve tumors.

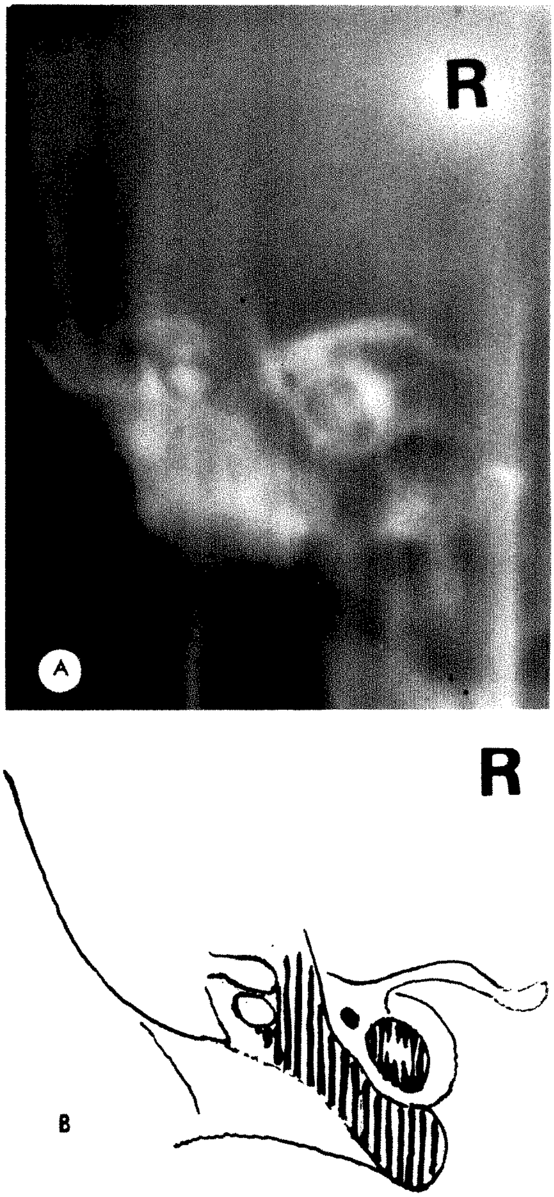

Fig. 10. (*A* and *B*) This patient with unilateral congenital deafness shows a round sac-like cochlea with absence of a normal spiral lamina. The facial canal can be seen above and lateral to the deformed cochlea. In addition, the vestibule and semicircular canals were absent and a deformed ossicular mass can be seen laterally. These findings were corroborated at operation.

MATERIAL

Initially, 50 children with congenital neurosensory deafness were examined by this method. Later, 7 parents were also studied. Of this group, 6 children and 1 adult were found to have roentgenographically recognizable bone abnormalities of different types. Involvement of the internal auditory canals was noted in several cases. Both unilateral and bilateral narrowing was seen. In these cases, the canals measured less than 1.5 mm. at their widest portion, a diameter which is below that considered normal (Fig. 7, *A* and *B*; and 8, *A* and *B*). The length of the canals was normal in these cases. Two cases were discovered with markedly shortened, wide canals with a depth of 4 mm. and width of 9 mm. (Fig. 9, *A* and *B*). We feel that purely on roentgenologic appearance these could represent an unusual variant of normal. However, the finding of congenital deafness in these patients as well as absence of this type of appearance in any of a large group of patients seen by us is highly suggestive that this is a significant abnormality of developmental origin.

Absence of the cochlea, previously reported by Everberg, was also found in 1 patient. In addition, a second patient showed a deformed cochlea (Fig. 10, *A* and *B*). The outer or first coil of the spiral lamina was present as a very thin bony circle and the remainder of the bony cochlea was not visualized roentgenographically. At surgery, a deformed saccular cochlea whose configuration matched the roentgenographic findings was found.

One of the most interesting types of anomaly in this group has also been previously reported by Everberg. This involves the bony vestibule and presents as marked enlargement with a diameter of up to 8 mm. The enlarged vestibule incorporates varying portions of the bony framework of the semicircular canals, particularly the horizontal (Fig. 8, *A* and *B*; and 11, *A* and *B*).

A similar abnormality has also been recently found in an 11 year old boy with unilateral deafness. Clinically and historically, the etiology could be on a congenital basis.

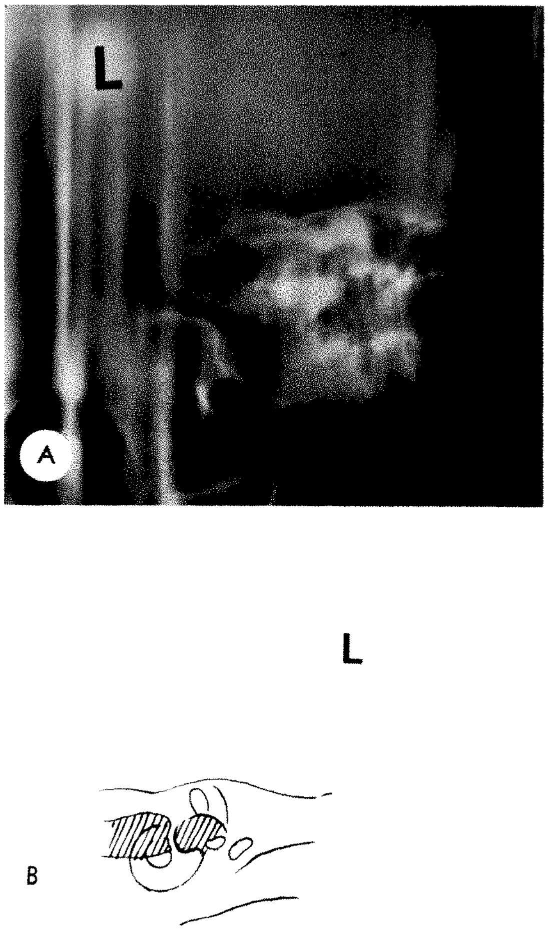

Fig. 11. (*A* and *B*) An additional case of unilateral congenital neurosensory deafness with marked enlargement of the vestibule and encroachment on the semicircular canals.

SUMMARY

The ability to determine fine anatomic detail in the temporal bone has been greatly enhanced by the use of plesiosectional tomography, a method readily available to many radiologists. The appearance of the temporal bone using this method in normal patients is described and changes found in patients with congenital neurosensory deafness are presented. The finding of bone abnormalities in this group has been of value to the otologists and audiologists handling this type problem and has given us considerable information about the roentgenographic appearance of the structure of the middle and inner ear.

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ATLAS-DENS INTERVAL (ADI) IN CHILDREN*

A SURVEY BASED ON 200 NORMAL CERVICAL SPINES

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IN JULY, 1964, an 8 year old girl was admitted to the State University of Iowa Hospitals with multiple injuries sustained in an automobile accident. Although she died soon after admission, numerous roentgenograms were obtained before death. A lateral roentgenogram of the cervical spine revealed a distance of 3.5 mm. between the anterior portion of the atlas and the dens (odontoid process) of the axis (Fig. 1). This raised the question of atlanto-axial dislocation. Autopsy was refused by the parents. A comparison of the atlas-dens interval (ADI) on roentgenograms taken before and after death revealed no change in the measurement. Stimulated by this finding, we measured the interval between the atlas and the dens in a normal 8 year old child and found the same distance, 3.5 mm. A hurried review of the literature revealed no adequate study of the ADI in children.[1–4] The present study was then initiated.

ANATOMY OF ATLANTO-AXIAL ARTICULATION

The first and second cervical vertebrae are functionally and anatomically different from the rest of the cervical spine. Flexion and extension of the head occur at the articulation between the occipital condyles and the facets of the atlas. No rotational movement occurs at this point. Movement occurring at the atlanto-axial articulation is primarily rotational (three-quarters of all rotational movement), although about 10 degrees of flexion can occur at this point.

Common anomalies of the dens or odontoid process which are confused with fractures are: failure of fusion of the summit epiphysis (appears at the age of 3 and fuses at the age of 12 years); failure of fusion between the body of the axis and the odontoid process; and agenesis of the odontoid process.

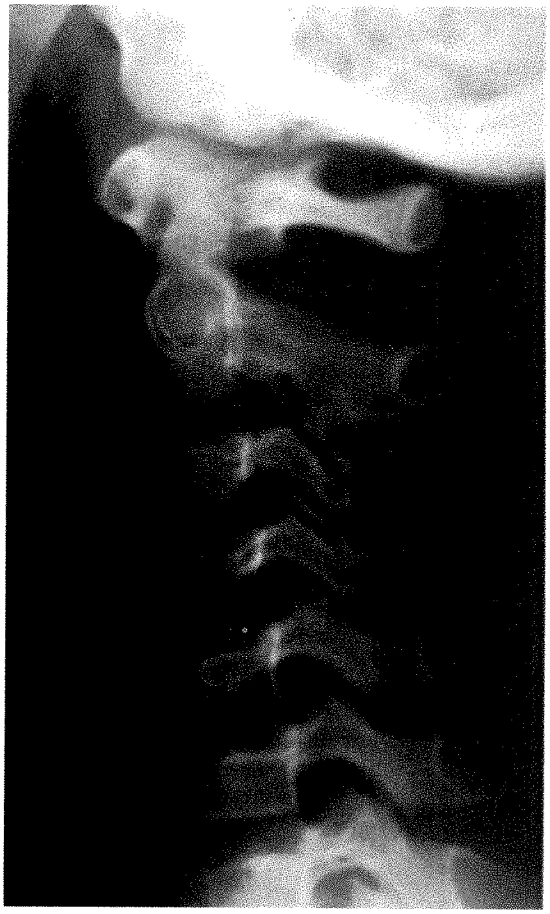

FIG. 1. Lateral cervical spine roentgenogram (40 inch tube to film distance) of an 8 year old female who was injured in an automobile accident. The atlas-dens interval (ADI) was initially interpreted as being increased.

A knowledge of the major ligaments of the atlanto-axial articulation is necessary to understand how difficult, if not impossible, it is for trauma to produce a subluxation at this level (Fig. 2). The major ligaments are: (1) The lateral alar-odon-

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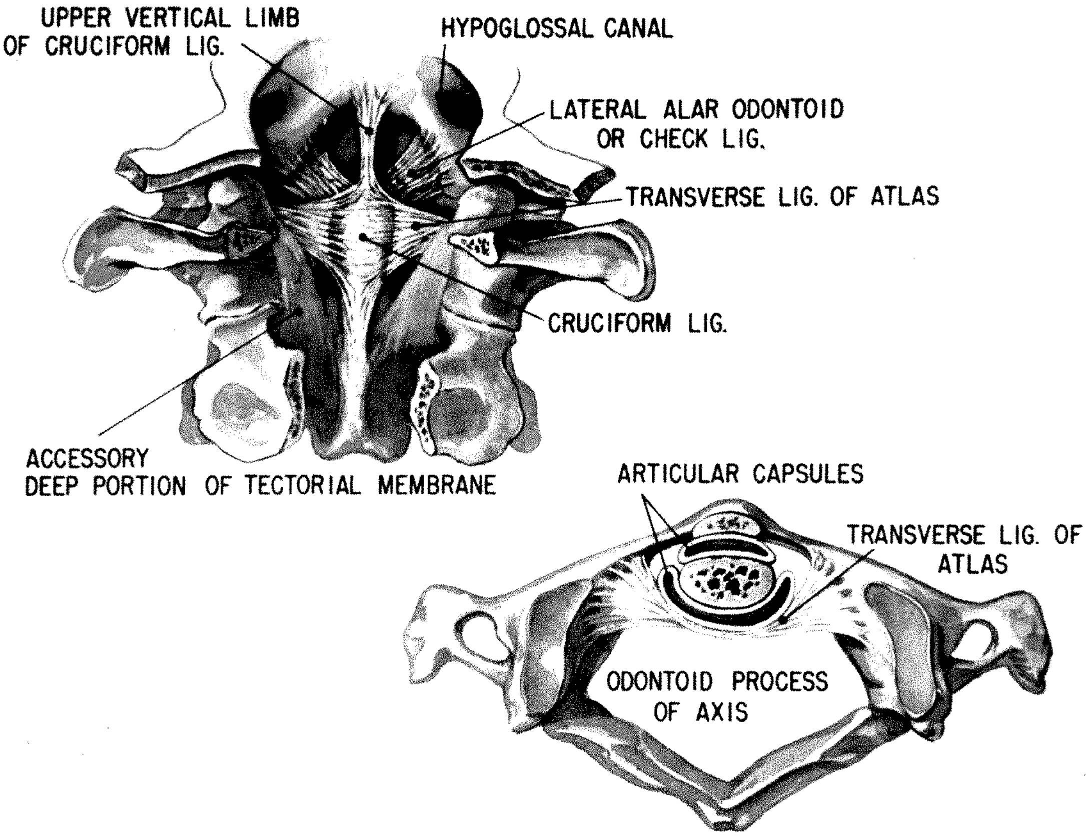

Fig. 2. The normal ligaments of the atlanto-axial articulation.

toid, or check ligaments, which extend from the medial aspect of the occipital condyles obliquely to the upper lateral aspect of the odontoid. Their purpose is to limit rotation of the skull. (2) The cruciate ligament consisting of the transverse ligament of the atlas which originates from the lateral masses of the atlas and crosses

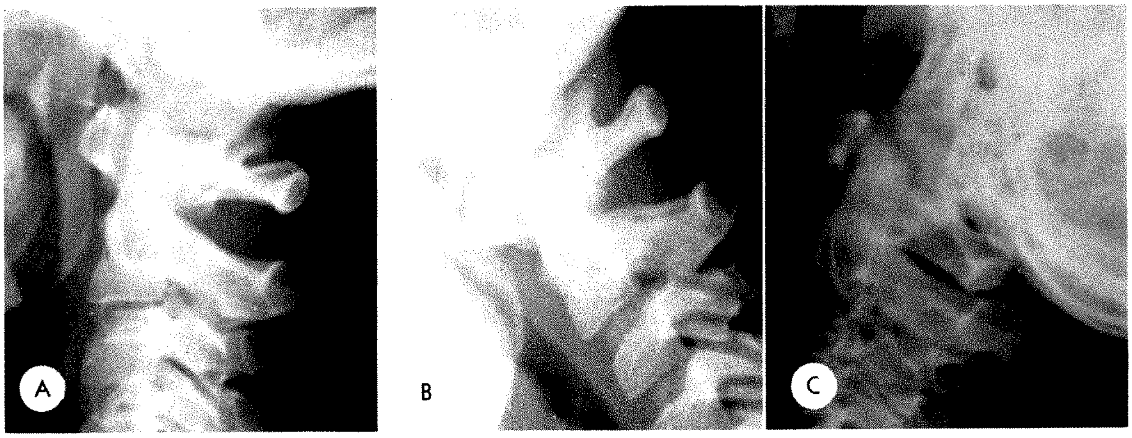

Fig. 3. (*A*, *B* and *C*) Lateral cervical spine roentgenograms (40 inch tube to film distance) of a normal 8 year old female in neutral, flexion and extension positions. The atlas-dens interval (ADI) is similar to that of the injured child.

behind the odontoid process. The cruciate ligament sends fibers superiorly to the anterior margin of the foramen magnum and inferiorly to the posterior surface of the body of the axis. When all other ligaments are severed, the transverse ligament will still hold the odontoid process close to the anterior arch of the atlas. Tremendous force is required to rupture the transverse ligament. Usually, atlanto-axial subluxation is accompanied by a fracture of the odontoid process, rather than a rupture of the transverse ligament.

PURPOSE AND METHODS

Lateral roentgenograms of the cervical spine in 200 normal children were analyzed for the following purposes: (1) to attempt to describe a frequency distribution of ADI measurements in normal children which could be used as a baseline to evaluate injured children; and (2) to see if sex, age, film to tube distance, position, and reader error had any significant effect upon ADI measurements. The age range was from 3 to 15 years. The lower limit of 3 years was selected because the summit epiphysis of the odontoid process is not present earlier, and adequate roentgenograms are more difficult to obtain in younger children. One hundred were done in the upright position using a 72 inch tube to film distance.

Another 100 studies were done at a 40 inch tube to film distance with the patient supine and films exposed with a horizontal beam. These positions were selected because the supine studies are usually utilized in trauma investigations, while children with inflammatory disease are examined upright. Lateral roentgenograms (Fig. 3, *A–C*), were taken in neutral position (*A*), flexion (*B*), and extension (*C*). The ADI was measured separately by two readers (L and G) on all roentgenograms. The interval measured was from the posterior inferior portion of the anterior arch of the atlas to the adjacent anterior border of the dens (Fig. 4). Neither reader watched the other measure the ADI. Calipers were used to transfer the measurement to an accurate 1 mm. ruler. Measurements were taken to the nearest 0.5 mm.

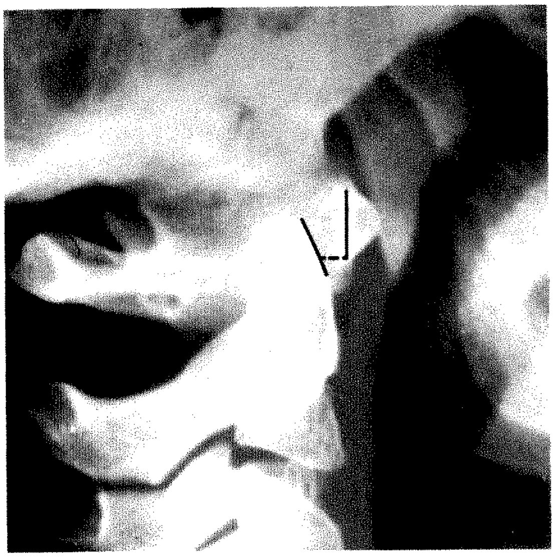

Fig. 4. A normal cervical spine showing the distance measured (broken line) between the posterior inferior portion of the anterior arch of the atlas and the anterior border of the dens.

RESULTS

The data were analyzed by the statistical department of our medical school. No significant difference was found for age (4 groups: 3–5 years, 6–8 years, 9–11 years, and 12–15 years) or sex. Histograms were used to demonstrate the frequency distribution of the ADI in this group of 200 children (Fig. 5, *A–D*). Changes in position, film to tube distance and variance in reader measurements interacted in such a way that specific conclusions regarding these factors are difficult to make. We are, however, justified in making the following statements: In the supine roentgenograms made with a 40 inch tube to film distance, no child had an ADI of more than 4 mm. Using the 72 inch tube to film distance and the upright neutral position, one child had an ADI of 5 mm. There was no history of trauma, arthritis or pharyngeal infection. This represents the largest ADI found in a normal child in any position.

The effects of change in position can be

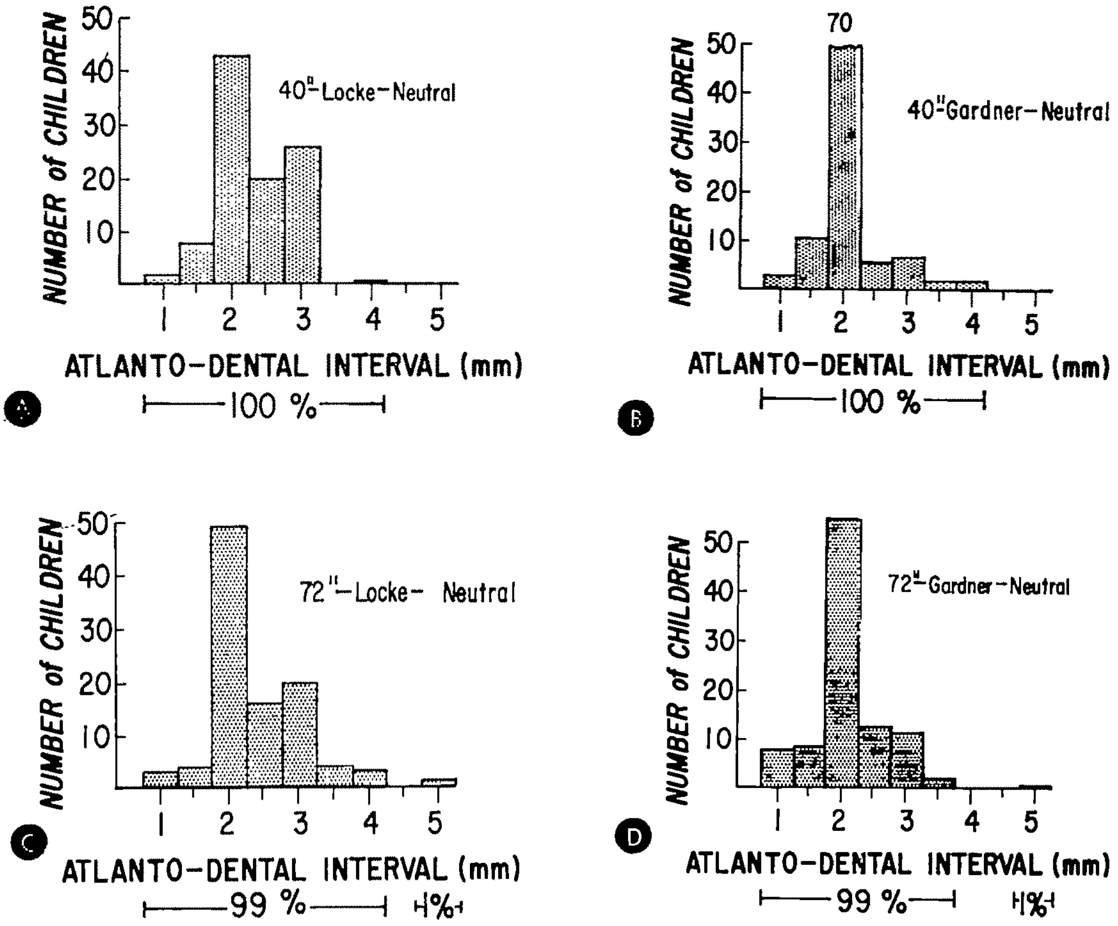

Fig. 5. (*A–D*) Histograms showing the distribution of the atlanto-dental interval (ADI) measurements in 200 normal children. One hundred children were examined with a 40 inch tube to film distance and a horizontal beam while in the supine position. One hundred other normal children were examined in the upright position with a 72 inch tube to film distance.

stated in general terms as follows: Flexion at both the 72 and the 40 inch tube to film distances tended to increase the ADI as did extension at the 40 inch tube to film distance for one of the readers (L). Extension at the 72 inch tube to film distance tended to decrease the interval. The changes in measurement are shown in Table I. It appears, therefore, that films should be exposed in neutral position.

Tube to film distance did not affect the ADI in the neutral and flexion positions. In the extension position, however, there was a statistically significant difference for both readers. It is our opinion that the magnification factor can be ruled out because of the neutral and flexion position results. The cause most likely was related to method of acquiring the cervical extension with the child supine. Clinically, this would not be a problem as extension studies are rarely obtained initially in trauma investigations.

In all combinations but one (40 inch tube to film distance with extension) one reader (L) tended to find higher values than the other (G). The mean difference was 0.25 mm. (Table II).

Atlanto-axial subluxation occurs in clinical conditions other than trauma.[5,6] The pharynx has lymphatic connections with the articulation between the atlas and the

Table I

VARIATION OF THE ATLAS-DENS INTERVAL (ADI) MEASUREMENT WITH CHANGE IN POSITION AND THE TUBE TO FILM DISTANCE AS MEASURED BY 2 DIFFERENT READERS

Distance (inches)	Reader	No. of Times a>b	No. of Times a<b	Sign* Test Result	Conclusion	
72	L	$\frac{a}{F}>\frac{b}{N}$ 38	16 $\frac{a}{F}<\frac{b}{N}$	P<.01	F>N	F>E
72	G	F>N 30	20	N.S.	F=N	
72	L	N>E 41	18	P<.01	N>E	
72	G	N>E 39	23	P<.05	N>E	
40	L	F>N 46	17	P<.01	F>N	F=E
40	G	F>N 21	10	N.S.	F=N	
40	L	N>E 16	49	P<.01	N<E	
40	G	N>E 6	56	P<.01	N<E	

* P<.01 = statistically significant difference at the .01 level.
P<.05 = statistically significant difference at the .05 level.
N.S. = no statistically significant difference.
N = neutral position; F = flexion; E = extension.

axis. Consequently, infections in the tonsils, middle ear, teeth and nose may drain into the cervical spine area. With such involvement, hyperemia occurs, especially at the attachments of the ligaments of the articulation. With hyperemia, bone is resorbed and the transverse ligament may pull loose from its attachment to the atlas. With excessive motion or minimal trauma, atlanto-axial subluxation may occur. When the cervical spine is involved by rheumatoid arthritis or Strümpell-Marie disease, hyperemia also occurs and a similar clinical subluxation may be demonstrated. The detachment of the transverse ligament may occur at any time during the course of the disease. It is important to note that there is no definite correlation between the amount of subluxation and the presence or absence of clinical symptoms or signs. Young children with rheumatic fever may also have a similar mechanism of subluxation.

Table II

DIFFERENCE BETWEEN MEASUREMENTS OF 2 DIFFERENT READERS

Distance (inches)	Position	L-G* (mm.)	Sign Test Result
72	N	+.26	P<.01
72	F	+.54	P<.01
72	E	+.18	P<.05
40	N	+.24	P<.01
40	F	+.44	P<.01
40	E	−.10	N.S.

* Difference between L and G readers. N = neutral position; F = flexion; E = extension.

Cases of subluxation of the atlas on the axis have been reported with minimal trauma when there is agenesis of the odontoid process. Usually, there is less damage to the spinal cord when the subluxation is accompanied by fracture or agenesis of the odontoid process. This is because most neural damage is done by the compression of the spinal cord between the posterior arch of the atlas and the odontoid process. With rotation of the cervical spine, apparent narrowing of the atlanto-vertebral foramen occurs. Consequently, erect anteroposterior positioning is of great importance.

SUMMARY AND CONCLUSIONS

The atlas-dens interval (ADI) was measured on lateral cervical spine roentgenograms in 200 normal children, ages 3 to 15 years. One hundred examinations were done at a 40 inch tube to film distance with the child supine, and another 100 at 72 inch tube to film distance with the child sitting.

Flexion, neutral and extension roentgenograms were obtained and analyzed.

1. At the 40 inch tube to film distance with the patient supine and the head in neutral position, no child in our series had an ADI greater than 4 mm. Therefore, a measurement greater than 4 mm. should alert one to the possibility of atlanto-axial subluxation.

2. At the 72 inch tube to film distance with the patient erect, there was a 5 mm. ADI in a normal 13 year old boy. This was our maximum normal ADI at this distance.

3. Age and sex are not statistically significant factors in evaluating the ADI; however, position can influence the measurements. Therefore, the neutral position is recommended for measurement.

4. The mean difference in measurements between readers was 0.25 mm. in neutral position.

5. Other signs of atlanto-axial subluxation are: (a) increased soft tissue anterior to cervical spine; (b) greater than 10 degree flexion between the atlas and axis; (c) compensatory curve of the lower cervical spine; and (d) narrowing of the atlanto-vertebral foramen.

The authors wish to thank Paul Leaverton, Ph.D., and Darrol Heggen, M.S., for the statistical analysis, and for their invaluable suggestions and interpretation of the data.

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NORMAL INTERPEDICULATE DISTANCES (MINIMUM AND MAXIMUM) IN CHILDREN AND ADULTS*

By VINCENT C. HINCK, M.D., WILLIAM M. CLARK, JR., M.D.,
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IN 1934 Elsberg and Dyke[2] published normal values for interpediculate distances as determined on roentgenograms of the adult human spine. Results were expressed as "usual upper limits" and "extreme upper limits" of normal but the method by which these limits were determined was not defined. Their use in diagnosis of intraspinal tumor was demonstrated.

In 1953 Landmesser and Heublein[6] prepared measurements of interpediculate distances using roentgenograms of selected children from the population of a home and hospital for crippled children. Data were published as ranges of normal without reference to mean values or variance.

In 1955 Simril and Thurston[9] published maximum and minimum normal interpediculate distances of infants and children. Their subjects were grouped according to "estimated physical age" by use of the Wetzel grid and their norms expressed as maximum and minimum for 3 age groups (1 day to 2 months; 2 months to 2 years; and 2 years to 12 years). Although they included minimum norms, they made no specific reference to their practical value nor did they describe the method of data analysis by which they arrived at values for maximum and minimum normal.

The following year Schwarz[7] published "extreme upper limits" of normal interpediculate distances in children and adults, adding measurements of the sacral spinal canal. To our knowledge, these have been the only attempts to establish normal ranges for interpediculate distances.

These pioneer studies have established the clinical value of the interpediculate measurements in the diagnosis of intraspinal tumors at all ages. There is need, however, for a more definitive delineation of the range of normal interpediculate distances, particularly with regard to the variability of their values in any given age group at the various spinal canal levels.

PURPOSE OF INVESTIGATION

The present investigation was undertaken to provide refined *maximum* normal interpediculate measurements for use in diagnosis of intraspinal tumor and *minimum* norms for recognition of spinal canal stenosis. Since use of interpediculate measurements in tumor diagnosis needs no endorsement, emphasis will be on diagnosis of stenosis.

METHOD

The roentgenographic files of the University of Oregon Medical School were systematically searched for anteroposterior spine roentgenograms of white patients. The roentgenograms were screened for readability and attempt was made to eliminate subjects with significant anomalies and other problems likely to influence growth and development. This selection was made to minimize bias introduced by use of a hospital population. Studies selected for measurement were classified by sex and by age to the nearest month. (Technique: target-film distance = 40 inches.) All measurements were made by one of the authors using calipers and were recorded to the nearest tenth mm. Interpediculate distance (hereinafter IPD) was considered the shortest measurement between the medial surfaces of the pedicles of a given vertebra.

IPD measurements were made on 474 roentgenograms, including 353 of children

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TABLE IA

MEAN INTERPEDICULATE DISTANCE (IPD) OF EACH VERTEBRA BY AGE

Male and Female Combined (mm.)
Age Group (yr.)

	3, 4, 5	6, 7, 8	9, 10	11, 12	13, 14	15, 16	17, 18	Adult
Age (yr.)	4.1	7.1	9.4	11.6	13.6	15.6	17.5	>18
Vertebra								
C3	23.9	26.0	26.4	26.2	27.2	27.1	27.7	28.0
4	24.9	26.8	26.9	27.0	28.3	28.2	28.7	28.8
5	25.3	27.2	27.1	27.4	28.6	28.6	29.1	29.4
6	25.3	27.6	27.0	27.3	28.5	28.2	29.1	29.3
7	24.4	26.4	26.2	25.9	27.2	26.9	27.6	28.0
T1	21.4	22.4	23.4	23.5	23.4	23.5	23.1	24.0
2	18.2	18.9	20.0	19.8	20.1	19.9	19.8	20.5
3	16.9	17.6	18.4	18.1	18.7	18.1	18.4	18.8
4	16.0	17.0	17.6	17.2	18.1	17.3	17.8	18.1
5	15.8	16.8	16.9	16.8	17.7	17.1	17.5	17.6
6	15.8	16.7	16.5	16.8	17.8	16.7	17.3	17.3
7	16.0	16.9	16.5	16.8	18.2	16.9	17.5	17.4
8	16.3	17.6	16.7	17.1	18.4	17.3	18.0	17.7
9	16.4	17.8	17.0	17.4	18.7	17.7	18.3	18.2
10	16.4	18.1	17.2	17.7	18.8	17.8	18.8	18.7
11	17.6	19.2	18.5	18.9	20.3	19.5	20.0	20.2
12	19.9	21.2	21.2	21.4	23.1	22.3	22.8	23.2
L1	20.5	21.8	23.0	23.0	23.7	23.9	24.5	25.0
2	20.4	21.9	23.2	23.1	23.4	23.9	24.4	25.5
3	20.8	22.4	23.8	23.6	24.0	24.6	24.8	26.0
4	21.7	23.3	24.8	24.9	25.7	25.6	26.1	26.9
5	24.5	26.5	28.3	28.7	29.1	29.1	29.6	29.7

(under the age of 19 years) and 121 of adults. The first 2 cervical vertebrae were excluded as unmeasurable and sacral measurements were omitted for want of an adequate sample. Since the roentgenograms had been taken for purposes other than this study, it was not possible in all cases to visualize the entire spine for measurement. Hence, the sample size varies from vertebra to vertebra. In general, the lumbar segment is most heavily represented and the cervical the least.

MEASUREMENT ERROR

Eighty-nine duplicate measurements were made, independently and at different times, in 9 different cases. The differences between duplicates ranged from zero to an extreme of 3.6 mm. The median absolute error was 0.26 mm. and, because of a few extremes, the mean difference was 0.40 mm. Of the 89 duplicates measured, 10 per cent had zero error, 50 per cent had less than 0.26 mm., 75 per cent had less than 0.50 mm. and 90 per cent had less than 1.0 mm. error.

RESULTS

IPD mean values and standard deviations were computed for each vertebral level separately by sex and for broad age groupings (3, 4, 5; 6, 7, 8; 9, 10; 11, 12; 13, 14; 15, 16; 17, 18 years and adult). Age group mean values are given disregarding sex in Table 1A, for males only in Table 1B and for females only in Table 1C. Standard deviations and sample sizes for each age group are given in Table II, male and fe-

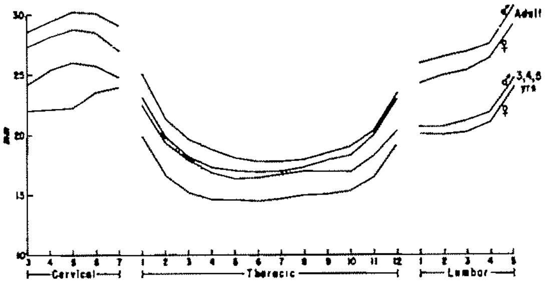


Fig. 1. Mean interpediculate distance (IPD), each vertebra, age 3, 4, 5 years and adult, by sex.

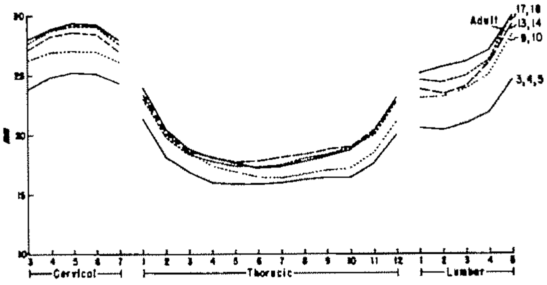


Fig. 2. Mean interpediculate distance (IPD), each vertebra, 5 age groups, sexes combined.

male combined, corresponding to the mean values in Table 1A.

The relative importance of age and sex in determining mean IPD can be seen in Table 1, or more easily in Figure 1, in which IPD mean values are plotted for each sex and for the two age extremes, 3, 4, 5 years and adult. It is evident that average male measurements are consistently larger than female by about 1 mm. The age difference is more striking, the growth being about 3 to 5 mm. from age 3, 4, 5 to adulthood. Some irregularities in the age-sex curves, *e.g.*, cervical for females age 3, 4, 5, are due

Table IB

MEAN INTERPEDICULATE DISTANCE (IPD) OF EACH VERTEBRA BY AGE

Male (mm.)
Age Group (yr.)

	3, 4, 5	6, 7, 8	9, 10	11, 12	13, 14	15, 16	17, 18	Adult
Age (yr.)	4.0	7.0	9.4	11.6	13.5	15.6	17.4	>18
Vertebra								
C3	24.3	26.3	26.9	26.0	26.9	27.9	28.6	28.6
4	25.5	27.4	27.3	27.8	28.0	28.8	29.5	29.5
5	26.1	27.8	27.5	27.0	28.4	29.3	29.9	30.3
6	25.8	28.2	27.3	27.2	28.4	29.2	30.1	30.2
7	24.8	27.1	26.2	26.0	27.3	28.0	28.8	29.3
T1	22.5	22.7	23.8	23.8	24.2	25.2	24.5	25.1
2	19.4	19.7	20.4	20.6	20.6	21.7	21.2	21.4
3	17.9	18.1	19.0	18.8	19.1	19.2	19.4	19.6
4	16.9	17.6	18.2	17.6	18.3	18.0	18.7	18.8
5	16.4	17.3	17.4	17.4	18.3	17.7	18.4	18.2
6	16.5	17.3	18.8	17.4	18.3	17.3	18.2	17.8
7	16.7	17.5	16.5	17.4	18.5	17.6	17.9	17.8
8	17.0	18.1	16.7	17.6	18.9	18.3	18.4	18.0
9	17.0	18.5	17.0	17.8	19.3	18.6	18.7	18.6
10	16.9	18.8	17.3	18.1	19.5	18.1	18.9	19.1
11	18.2	20.1	18.8	19.2	20.9	19.8	20.2	20.4
12	20.4	22.5	21.3	21.8	23.5	23.2	23.4	23.5
L1	20.7	22.5	23.3	23.9	23.8	24.5	25.1	25.9
2	20.7	22.4	23.5	24.2	23.3	24.6	24.8	26.5
3	21.2	23.0	24.1	24.6	23.6	25.1	25.2	26.8
4	21.9	23.6	24.8	23.6	24.7	26.0	26.6	27.6
5	24.7	26.9	28.4	28.9	28.0	30.1	29.9	30.7

Table IC

MEAN INTERPEDICULATE DISTANCE (IPD) OF EACH VERTEBRA BY AGE

Female (mm.)
Age Group (yr.)

	3, 4, 5	6, 7, 8	9, 10	11, 12	13, 14	15, 16	17, 18	Adult
Age (yr.)	4.2	7.2	9.6	11.6	13.7	15.5	17.6	>18
Vertebra								
C3	22.1	25.5	24.8	26.6	27.5	26.6	26.6	27.4
4	22.2	26.0	25.6	27.4	28.4	27.7	27.5	28.2
5	22.3	26.4	25.9	28.0	28.7	28.1	27.9	28.7
6	23.6	26.7	26.0	27.3	28.6	27.5	28.0	28.6
7	24.0	25.5	26.3	25.8	27.1	26.1	26.1	27.1
T1	19.8	22.1	23.1	23.0	22.6	22.3	22.4	23.1
2	16.6	18.2	19.7	18.8	19.6	18.9	19.1	19.8
3	15.2	17.0	17.9	17.5	18.3	17.6	17.9	18.2
4	14.6	16.3	17.1	16.6	17.8	16.9	17.4	17.4
5	14.6	16.3	16.6	16.0	17.2	16.8	17.1	17.1
6	14.5	16.1	16.3	15.9	17.4	16.5	16.9	16.9
7	14.7	16.3	16.5	16.1	17.8	16.6	17.4	17.0
8	15.0	16.9	16.6	16.4	18.0	16.8	17.8	17.4
9	15.1	17.1	16.9	16.8	18.1	17.3	18.2	17.9
10	15.4	17.3	17.1	17.1	18.2	17.7	18.7	18.4
11	16.5	18.0	18.2	18.4	19.8	19.2	19.9	20.0
12	19.1	20.2	21.1	20.9	22.8	21.7	22.5	22.9
L1	20.1	21.0	22.5	22.2	23.7	23.6	24.1	24.3
2	20.0	21.1	22.6	22.3	23.6	23.6	24.2	24.9
3	20.2	21.7	23.2	22.8	24.6	24.4	24.5	25.4
4	21.1	23.0	24.7	24.2	26.9	25.4	25.8	26.4
5	23.9	26.1	28.2	28.5	30.4	28.6	29.5	29.0

to different sample sizes obtained in this refined breakdown. Since the sex effect is an order of magnitude less than that of age or clinical significance, it was decided to present the "normal" tolerance ranges irrespective of sex.

The effect of age, with sexes combined, can be seen in Figure 2. The mean values for ages 3, 4, 5; 9, 10; 13, 14; 17, 18 years and adult are plotted. There is a steady progression of mean IPD of all levels with age. In the cervical segment there is a substantial increase from age 3, 4, 5 to age 9, 10 (about 2 mm.) and another increase (1.5 mm.) to age 13, 14; thereafter, there is slight increase. In the thoracic segment the main growth again takes place in the early years from 3, 4, 5 to 9, 10 and this early growth is still more marked in the lumbar segment, the average increase from the 3, 4, 5 to 9, 10 age group being nearly 3 mm.

The interperson variability, represented by the standard deviation (Table II), is reasonably constant for age and sex but varies considerably by vertebra. The cervical vertebrae are about equally variable, having standard deviations of 1.4 to 2.8 mm. There appears to be a slight increase in variability in the upper ages, attributable perhaps, in part, to the increased difficulty of visualization, as well as to the greater differences in general somatic size. The age group 11, 12 years was consistently more variable than any other age. This increased variability comes mainly from males who have standard deviations of 3 mm. and more in this age group. Since this is the period of onset of the skeletal growth spurt for some boys, it is assumed that the increased variability is reflecting individual differences in the timing of this growth.

Interperson variability in the thoracic segment is least in the vertebrae with smaller IPD measurements (3–10) and uniform across the ages, with the exception of a slight increase in variability in the 13, 14 age group, related most probably to individual differences in growth at this period. The vertebrae with larger measurements (1, 2, 11, 12) are slightly more variable.

In the lumbar segment, there is a slight trend of increasing variability with age, but the most striking feature is a uniformly greater variability in L4 and 5 than in 1, 2 and 3. This is due to the fact that although the IPD's of L4 and 5 are generally larger than those of L1, 2, and 3, in a few instances they are actually smaller.

Norms for use in clinical appraisal have been constructed by computing, for each vertebra and each age group, 90 per cent tolerance ranges based on the combined male and female samples (Table III, Fig. 3, *A–H*). These tolerance ranges are the low and high limits within which the central 90 per cent of "normals" may be expected to fall:[1] Only 5 per cent of "normals" should be below the low limit and only 5 per cent above the high limit. The width of these ranges varies somewhat from age group to age group and from vertebra to vertebra because of 2 relevant factors: the

Table II

STANDARD DEVIATION OF INTERPEDICULATE DISTANCE (IPD) OF EACH VERTEBRA BY AGE

Male and Female Combined (mm.)
Age Group (yr.)

	3, 4, 5	6, 7, 8	9, 10	11, 12	13, 14	15, 16	17, 18	Adult
		Standard Deviation in mm. (Sample Size, N)						
Age (yr.)	.09 (35)	.08 (52)	.05 (30)	.05 (46)	.05 (59)	.05 (63)	.05 (68)	— (121)
Vertebra								
C3	1.7 (8)	1.5 (18)	1.7 (8)	2.6 (18)	1.2 (22)	1.8 (17)	1.9 (21)	1.5 (36)
4	1.7 (8)	1.5 (18)	1.3 (8)	2.5 (18)	1.5 (23)	1.6 (18)	2.0 (22)	1.5 (43)
5	1.7 (8)	1.7 (18)	1.6 (8)	2.6 (19)	1.7 (23)	1.6 (18)	2.1 (22)	1.7 (44)
6	1.7 (9)	1.7 (18)	1.7 (8)	2.7 (20)	2.1 (23)	1.8 (20)	2.2 (24)	1.8 (50)
7	1.4 (9)	2.0 (19)	2.1 (8)	2.8 (21)	1.8 (25)	2.4 (21)	2.1 (24)	2.0 (60)
T1	2.5 (12)	1.9 (14)	1.4 (10)	2.2 (22)	1.5 (17)	2.3 (19)	1.6 (27)	1.8 (53)
2	2.2 (12)	1.6 (14)	1.2 (10)	2.0 (23)	1.8 (17)	2.4 (21)	1.4 (27)	1.7 (53)
3	1.6 (12)	1.5 (14)	0.9 (9)	1.6 (23)	1.6 (17)	1.5 (20)	1.3 (27)	1.5 (50)
4	1.5 (12)	1.4 (14)	1.0 (9)	1.7 (23)	1.8 (15)	1.3 (19)	1.3 (26)	1.6 (43)
5	1.4 (13)	1.2 (14)	0.8 (9)	1.6 (22)	1.8 (15)	1.4 (19)	1.3 (25)	1.7 (42)
6	1.6 (13)	1.2 (13)	1.0 (9)	1.5 (22)	1.8 (15)	1.3 (19)	1.6 (25)	1.5 (42)
7	1.6 (12)	1.2 (13)	1.3 (9)	1.4 (22)	1.9 (15)	1.6 (19)	1.5 (26)	1.5 (42)
8	1.6 (13)	1.4 (14)	1.0 (9)	1.5 (22)	1.9 (15)	1.6 (19)	1.5 (25)	1.5 (41)
9	1.5 (13)	1.3 (15)	1.3 (10)	1.6 (23)	1.8 (15)	1.8 (19)	1.6 (26)	1.5 (43)
10	1.3 (14)	1.4 (17)	1.6 (10)	1.7 (23)	2.0 (17)	1.6 (20)	1.5 (26)	1.6 (44)
11	1.5 (15)	1.9 (17)	1.8 (10)	1.8 (23)	2.3 (18)	1.7 (24)	1.7 (30)	1.8 (53)
12	1.5 (17)	2.1 (17)	1.6 (11)	1.8 (24)	2.3 (20)	1.7 (28)	1.7 (31)	2.0 (59)
L1	1.6 (26)	1.9 (33)	1.8 (20)	1.7 (26)	1.6 (35)	2.0 (40)	2.4 (37)	2.2 (59)
2	1.5 (26)	2.0 (32)	1.6 (20)	1.8 (25)	1.6 (35)	1.8 (40)	2.2 (36)	2.3 (57)
3	1.6 (26)	2.1 (33)	1.7 (20)	1.9 (25)	2.0 (35)	1.9 (40)	2.3 (36)	2.7 (53)
4	1.7 (26)	2.1 (33)	2.3 (20)	3.1 (24)	3.3 (34)	2.4 (39)	2.9 (36)	3.0 (52)
5	1.9 (26)	3.1 (33)	2.7 (20)	3.1 (23)	3.6 (34)	2.9 (38)	3.6 (35)	3.7 (50)

TABLE III

TOLERANCE RANGE (90 PER CENT) OF INTERPEDICULATE DISTANCE (IPD) OF EACH VERTEBRA BY AGE

Male and Female Combined (*mm.*)
Age Group (*yr.*)

	3, 4, 5	6, 7, 8	9, 10	11, 12	13, 14	15, 16	17, 18	Adult
Vertebra								
C3	18–29	22–30	21–32	20–32	24–31	23–31	23–32	25–31
4	19–30	23–31	21–32	21–33	25–32	24–32	24–33	26–32
5	20–31	23–31	22–32	21–33	25–32	25–32	25–34	26–33
6	20–31	24–31	22–32	21–33	25–32	24–33	25–34	26–33
7	19–30	23–31	21–32	20–32	24–31	21–32	23–32	24–32
T1	17–26	19–26	20–27	20–27	19–28	18–29	20–26	20–28
2	14–22	15–22	17–24	16–24	16–24	14–25	17–23	17–24
3	13–21	14–21	15–21	14–22	15–23	15–22	15–21	16–22
4	12–20	14–21	15–21	14–21	14–22	14–20	15–21	15–21
5	12–20	13–20	14–20	13–21	14–22	14–21	15–21	14–21
6	12–20	13–20	14–20	13–20	14–22	13–20	14–20	14–20
7	12–20	13–21	14–20	13–20	14–22	13–21	15–21	14–20
8	12–21	14–21	14–20	13–21	14–23	14–21	15–21	15–21
9	12–21	14–21	13–21	14–21	15–23	14–22	15–21	15–21
10	12–21	15–22	13–21	14–21	15–23	14–22	16–22	16–22
11	13–22	16–23	14–23	15–22	16–25	16–23	17–23	17–24
12	16–24	18–25	17–25	18–25	19–27	18–26	20–26	19–27
L1	17–24	17–27	19–28	19–27	20–27	20–28	20–29	21–29
2	17–24	17–27	19–28	19–27	20–27	20–28	20–29	21–30
3	17–24	17–27	19–28	20–27	21–28	21–29	20–29	21–31
4	18–25	18–28	20–29	20–28	19–33	21–30	19–33	21–33
5	21–28	22–32	24–33	24–34	22–36	23–35	23–37	23–37

intrinsic interperson variability (estimated by the standard deviation of the observed sample) and the sample size on which our estimates of mean and variability are based. For example, the limits for L4 and L5 at age 13, 14 are quite far apart because the variation in our sample was large and, in addition, our sample sizes were smaller.

Averages of sample sizes and standard deviations of groups of vertebrae that were similar were used in computing tolerance limits by adding to and subtracting from the mean 2.8, 2.5, 2.3 and 2.1 standard deviations for estimates based on sample sizes of 10, 15, 20 and 30, respectively, following the probability tables given by Dixon and Massey.[1]

The tolerance ranges are recommended for clinical use as an index of suspicion of abnormality. Any carefully measured individual IPD falling outside the given limits should be viewed with suspicion of pathology or anomaly, though these limits are surely not stringent enough to detect all anomalies. Similarly, failure to exceed the limits cannot guarantee normalcy.

All normal spines tend to exhibit a similar profile of the 22 vertebral IPD's, individual differences being more in the overall size level than in the sizes of single vertebrae. Thus, a case in which most of the vertebrae have IPD's close to the mean value but a single vertebra or adjacent pair of vertebrae is distinctly out of line should be viewed with suspicion even though none of the individual values exceeds the tolerance limits. This type of abnormality may be suspected by comparing the *shape* of the patient's IPD curve with the normal.

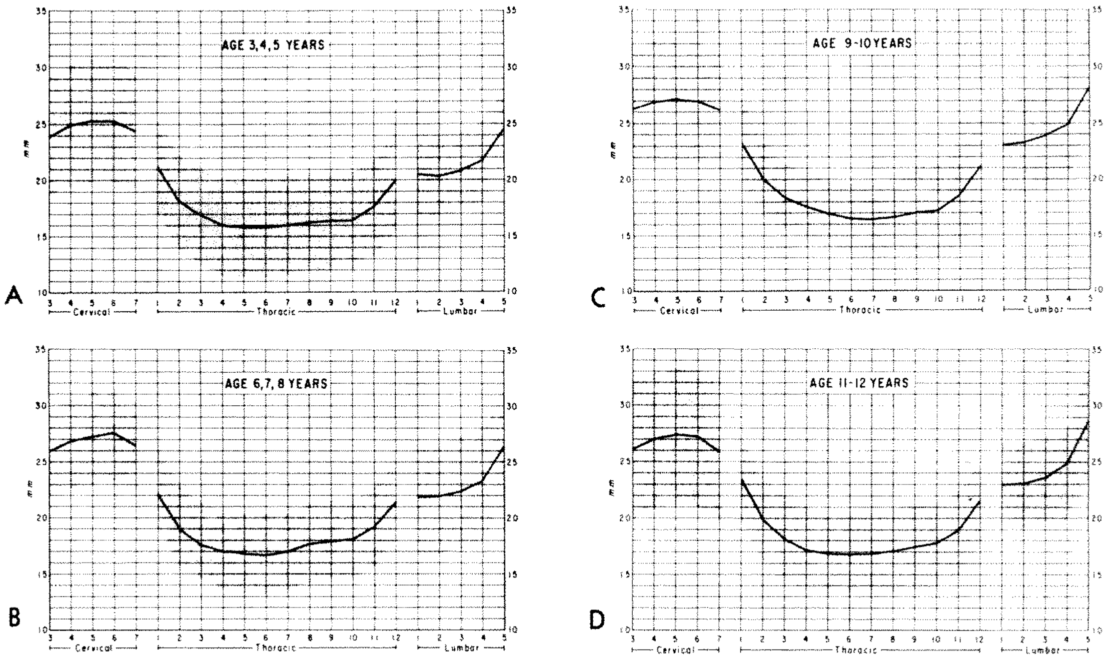


FIG. 3. Tolerance range (90 per cent) of interpediculate distance (IPD), each vertebra, male and female combined. (*A*) Age 3, 4 and 5 years. (*B*) Age 6, 7 and 8 years. (*C*) Age 9 and 10 years. (*D*) Age 11 and 12 years.

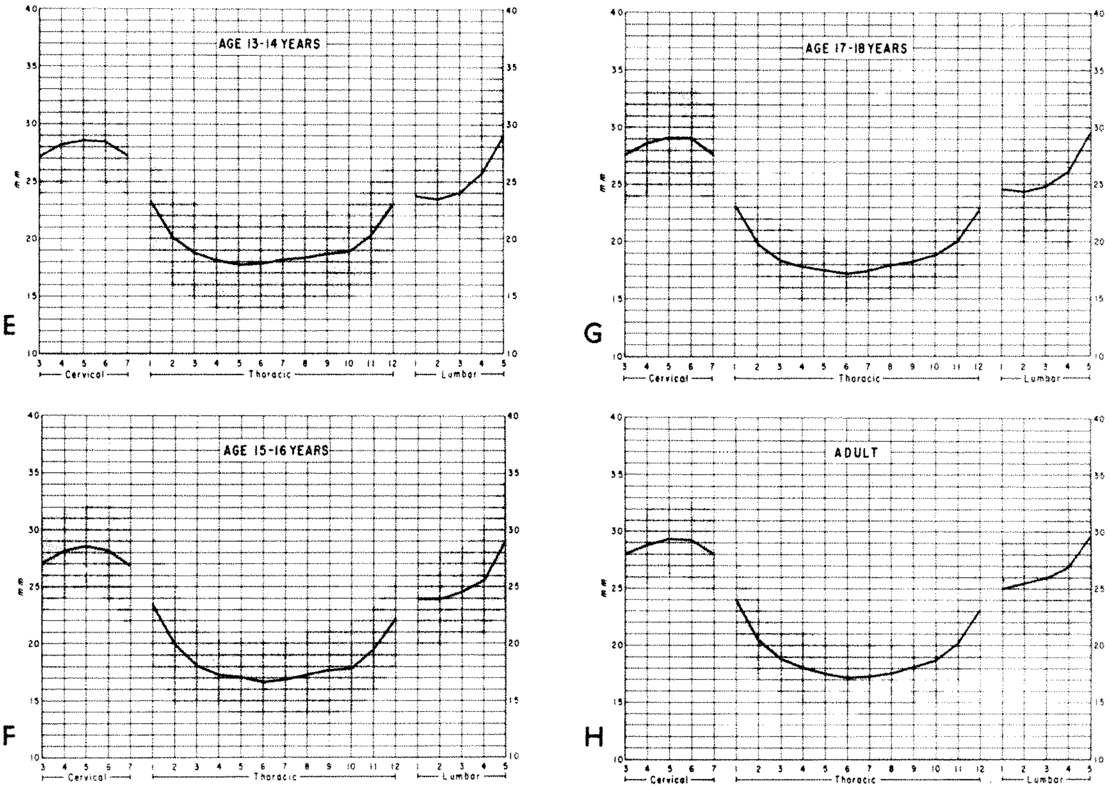


FIG. 3. Tolerance range (90 per cent) of interpediculate distance (IPD), each vertebra, male and female combined. (*E*) Age 13 and 14 years. (*F*) Age 15 and 16 years. (*G*) Age 17 and 18 years. (*H*) Adult.

TABLE IV

ANTEROPOSTERIOR SPINE IPD—POSTEROANTERIOR CHEST IPD

Mean Difference at Each Vertebral Level (mm.)

Vertebra	N	Difference
T1	8	1.46
2	8	1.06
3	8	0.79
4	9	0.58
5	10	0.38
6	9	0.08
7	8	0.31
8	7	0.64
9	5	0.22
10	8	0.20
11	6	0.58
12	3	0.43

IPD: ANTEROPOSTERIOR SPINE VERSUS POSTEROANTERIOR CHEST ROENTGENOGRAMS

In clinical practice, the question occasionally arises whether IPD measurements from a posteroanterior chest roentgenogram can be compared with normal standards derived from anteroposterior spine roentgenograms. Consensus seems to have been that the 2 cannot be used interchangeably because the IPD from the chest film would be considerably magnified. To the best of our knowledge, however, this is an opinion without documentation.

Accordingly, we undertook a comparison of the 2 measurements by obtaining both posteroanterior chest and anteroposterior thoracic spine roentgenograms (target-film distances 72 inches and 40 inches, respectively) of each of 10 randomly selected normal patients between 10 and 15 years of age. IPD measurements were made on both roentgenograms in the usual manner. Due to obscurity of some vertebrae on several chest roentgenograms, there were 89 comparable measurements instead of 120. To our surprise IPD measurements from the spine roentgenograms were *larger* than those from the chest roentgenograms. (Silverman[8] independently has made this observation.) On the average the difference was about 0.6 mm.

The data were further analyzed to determine whether there was local variation in this difference. The mean difference between the 2 measurements was determined for each vertebra. The results are shown in Table IV and illustrated in Figure 4. It will be noted that there *are* local variations of the mean difference; the difference is more striking when values for T1–3 (mean difference of 1.10 mm.) are compared with those of T4–12 (mean difference of 0.38 mm.).

It may be that variations of IPD difference at these 2 levels are due to differences of relationship between spine and cassette in the 2 projections. When the individual is positioned for an anteroposterior recumbent thoracic spine roentgenogram, the vertebral column tends to straighten but still retains some of its kyphotic curve. T1

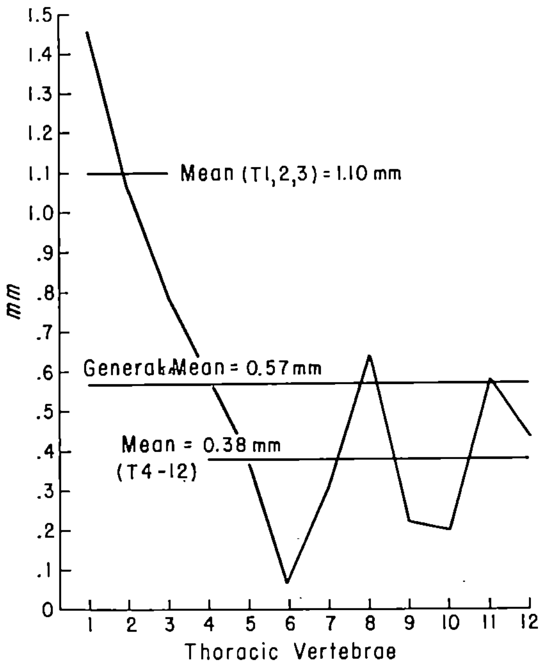


FIG. 4. Mean difference between thoracic IPD measurements from anteroposterior thoracic spine roentgenograms and those from posteroanterior chest roentgenograms; recorded for individual vertebrae, for subdivisions of the thoracic spine, and for the thoracic spine generally.

is farther from the film than T6 and its IPD is more magnified than that of T6. Conversely, when positioned for an erect chest roentgenogram, the shoulders are hunched forward toward the cassette and T1 is closest to the cassette; T6 is farthest away.

Since anteroposterior measurements are generally greater than posteroanterior measurements (*i.e.*, more magnified) and T1 is the most magnified vertebra in the anteroposterior roentgenogram and the least magnified in the posteroanterior roentgenogram, it follows that the anteroposterior-posteroanterior difference should be larger for T1 than for the other thoracic vertebrae. Conversely, the anteroposterior-posteroanterior difference for T6 should be the smallest. Vertebrae between T1 and T6 should show a gradient of diminishing difference. Below T6, where the spine curve reverses and enters the "small of the back," the trend can be expected mildly to reverse. The curve of mean differences (anteroposterior spine–posteroanterior chest, Fig. 4) does indeed reflect these expected variations.

Though chest roentgenograms are *not* recommended for IPD determinations, it would appear from the foregoing analysis that they can be used if necessary with moderate accuracy, particularly for IPD's below T3.

DISCUSSION

Stenosis of the spinal canal due to decreased IPD is, to the best of our knowledge, virtually unexplored. Stenosis due to decreased sagittal diameter has been reported in the cervical spine[5] as well as the lumbar spine.[3,4,10] In either instance the anomaly may be clinically significant.

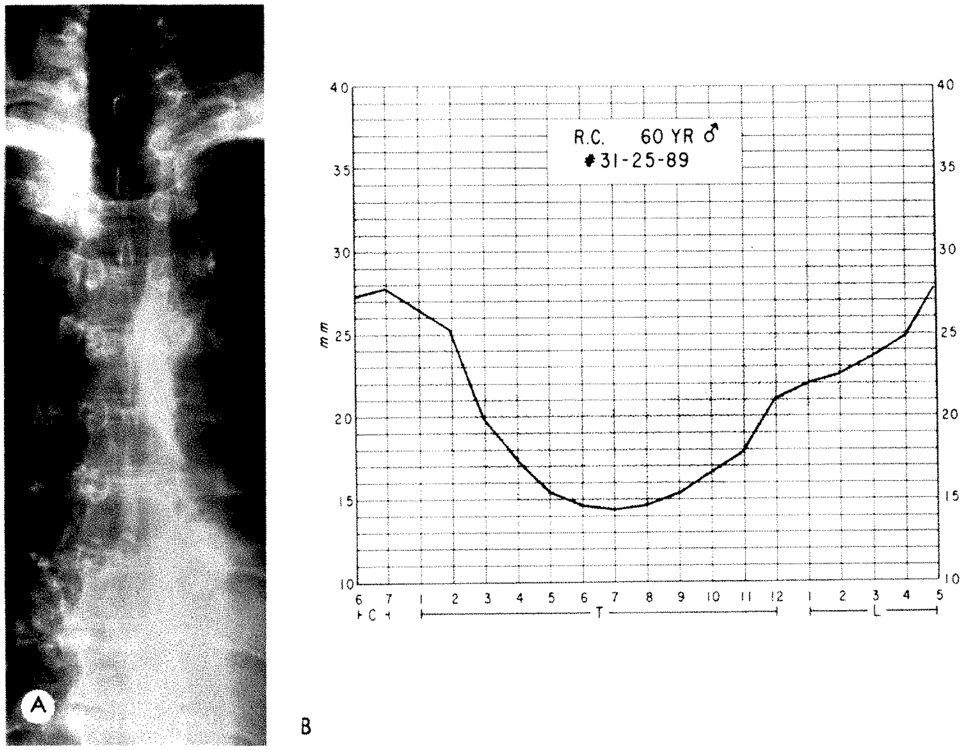
We undertook the determination of normal minimum IPD standards as a preliminary to clinical investigation of transverse spinal canal stenosis, its frequency and its significance. Our clinical experience is still limited but we can already state that (a) there is such an entity as developmental stenosis of the spinal canal with decreased IPD, (b) it may, but need not be, associated with decreased sagittal diameter, and (c) it may be symptomatic. By way of illustration the following cases are presented.

ILLUSTRATIVE CASES

Case 1. R.C. was a 60 year old male. Eleven months before admission, he first noted abdominal swelling which persisted until the time of admission. Six months before admission, he developed severe lancinating pains in the right upper quadrant and similar pains shooting into both legs. The pains lasted several seconds and then disappeared completely. They occurred as often as 10 times a day, in any position, day or night. He was studied at another hospital where he was thought to have a "right flank hernia" and was found to have diabetes mellitus that was easily controlled by appropriate diet. The symptoms continued. Three months before admission, unsteadiness of gait and weakness of legs developed, causing difficulty in climbing stairs. He also complained of mild paresthesias of the soles of the feet.

On examination, weak and flaccid abdominal musculature was found. This accounted for the apparent abdominal enlargement. Reflexes in the upper extremities were normal. Deep tendon reflexes were absent at both ankles and the right knee; and reduced at the left knee. Babinski responses were flexor. Cremasteric reflex was absent on the right. Abdominal reflexes were absent. Romberg's sign was negative. Position and vibratory sensations were moderately decreased in the lower extremities. There was patchy and variable decrease in temperature, light touch and superficial pain sensations in the lower extremities. Examination of the spinal fluid was normal except for a protein of 84 mg. per cent.

Roentgen Findings. Thoracic spine roentgenograms showed mild degenerative changes in the middle and lower thoracic spine and a small posterior osteophyte at T7–8. Although there were no available standards for minimal IPD's, it was believed that there was, at least, a relative stenosis of the mid-thoracic spinal canal. Due to minimal rotation of the lateral roentgenogram, the sagittal diameter could not be viewed. Morphology of vertebrae was otherwise unremarkable. The IPD measurements have subsequently been compared with our normal standards and are shown in Figure 5 *B*



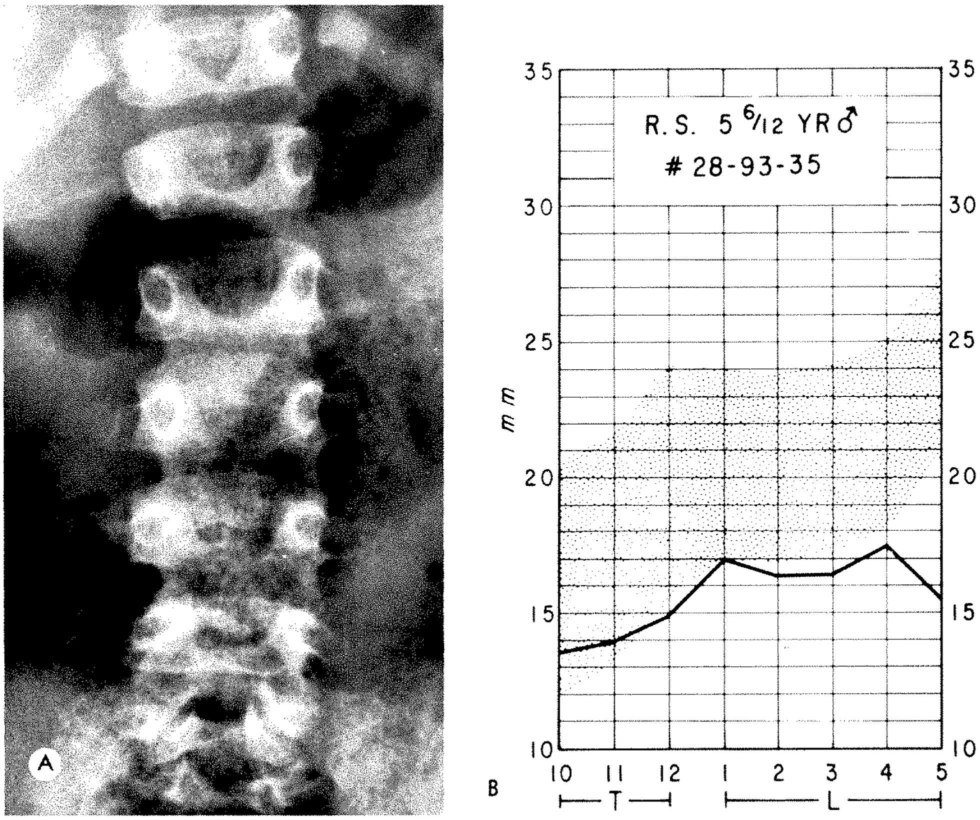


Fig. 6. Case 11. (*A*) Diminished IPD's, more so in the lower lumbar and sacral spinal canal. (*B*) Graph depicts patient's measurements plotted against 90 per cent tolerance range for his age group.

Fig. 5. Case i. (*A*) Mild degenerative changes in middle and lower thoracic spine. Mid-thoracic interpediculate distances appear small. Vertebral morphology is otherwise normal. (*B*) Graph compares patient's IPD's with 90 per cent tolerance range of normal. There is stenosis of the spinal canal, especially between T5 and T10. T7 and T8 are at the lower limit of the tolerance range.

←⋘

along with the anteroposterior spine roentgenogram (Fig. 5*A*).

At myelography, the contrast column moved freely to the upper level of T12 which it passed only after an appreciable interval of arrest. Despite a steep head-down table tilt, the remainder of the transit through the thoracic spinal canal was very slow and hesitant. After the contrast medium had accumulated at the cervical level, the table was tilted to a near-erect position. The column travelled caudad with ease as far as T3, where it gradually narrowed and again began a slow movement through the rest of the thoracic canal. The spinal cord appeared to narrow excessively as it entered the region of stenosis. No defect was reported at T7–8 but a persistent extradural defect was noted at T12–L1 on the left side. A lateral spot roentgenogram showed a ventral impression of the column at the same level.

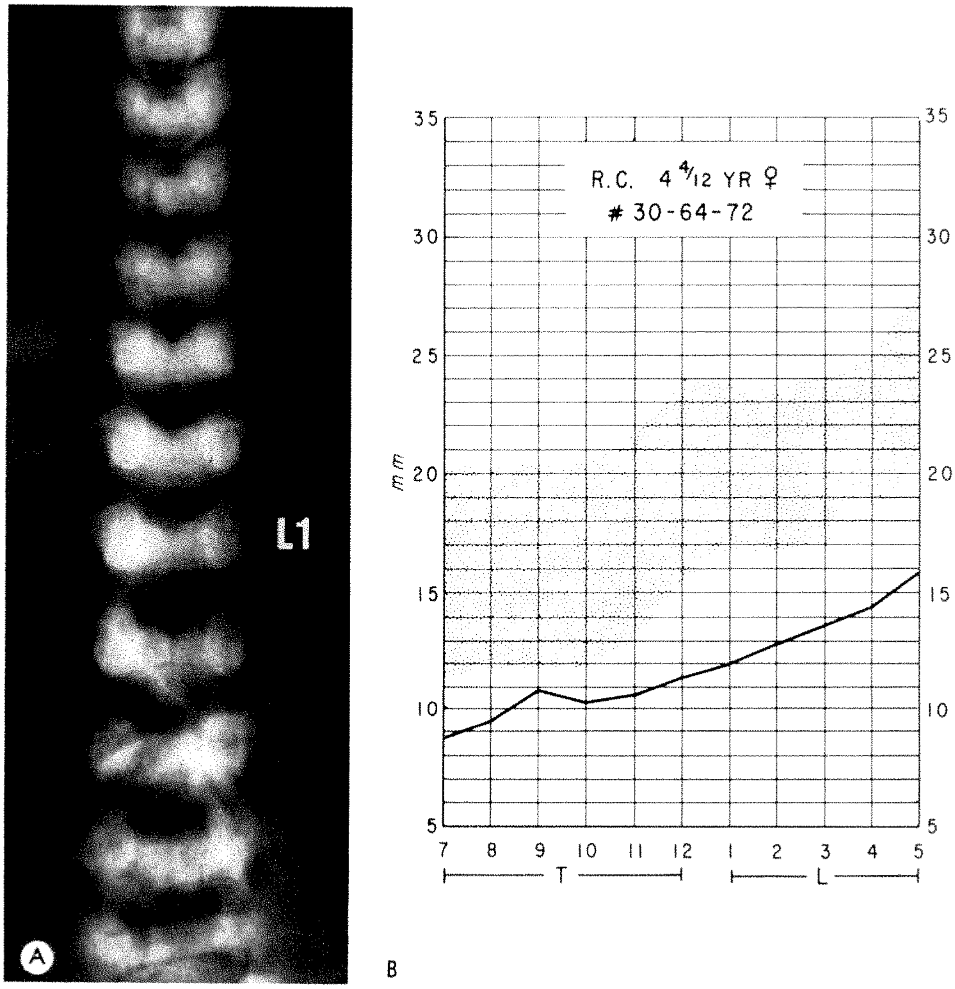


Fig. 7. Case iii. (*A*) Massive vertebral bone structure due to osteopetrosis. Large pedicles have narrowed the IPD's. (*B*) Graph depicts patient's measurements and the 90 per cent tolerance range for her age group.

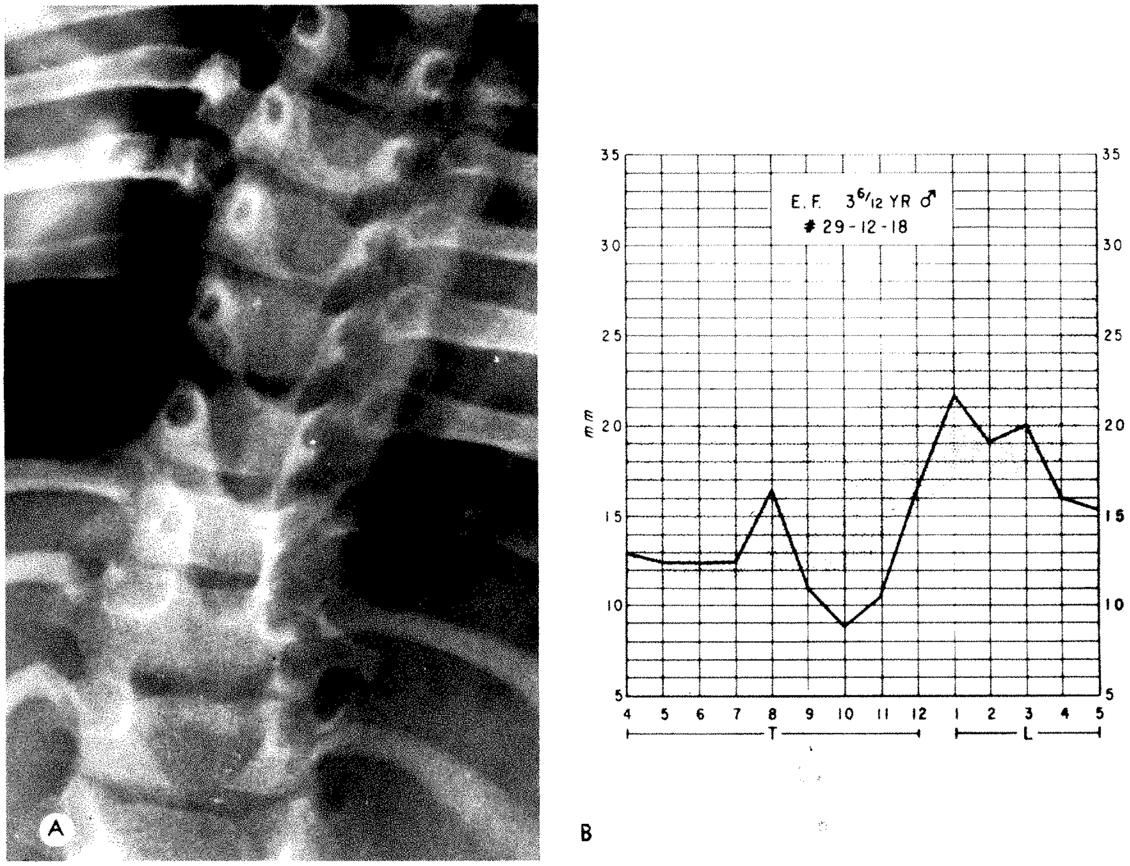


Fig. 8. Case iv. (*A*) Gross decrease of IPD measurements in lower thoracic spine. (*B*) Graph depicts patient's measurements and the 90 per cent tolerance range for his age group. Note variable size of vertebral bodies.

It was felt that the impression of thoracic spinal canal stenosis which had been gleaned from the conventional spine roentgenograms was amply supported both by the unusual dynamics seen at fluoroscopy and the unusual findings demonstrated on spot roentgenograms.

Comment. This patient demonstrated cord dysfunction roughly corresponding to the level of stenosis. It is suggested that he became symptomatic because degenerative changes developed in the thoracic spine leading to encroachment on the already narrow spinal canal and consequent spinal cord compression.

Despite lack of surgical confirmation, the presence of stenosis seems well established. The fact that the smallest IPD's are at, but not below, the tolerance limits does not militate against a diagnosis of stenosis. As stated previously in this paper, failure to exceed the limits cannot guarantee normalcy. The distribution curve of IPD measurements of patients with stenosis is not known, but evidently overlaps the distribution for normals. This patient's measurements fall within that area.

The following cases demonstrate more striking stenosis than Case i. Nonetheless, these children were asymptomatic. The lack of clinical manifestations probably depends on the greater ability of immature nervous tissue to compensate during the phase of active growth and maturation.

Case ii. R.S., a 5½ year old male achondroplastic dwarf, was referred to this hospital for evaluation of mental retardation. He had no signs, symptoms or neurologic findings which might be ascribed to lumbar spinal canal stenosis. Anteroposterior lumbar spine roentgenograms showed IPD's below T10 generally to be at or beyond the lower end of the 90 per cent tolerance range (Fig. 6, *A* and *B*). Stenosis was greatest at lower lumbar and sacral levels. Sagittal diameters of the spinal canal were also diminished.

Case iii. R.C., a 4 year old female, suffered from osteopetrosis. She was found to have stenosis of her spinal canal on roentgenologic examination, although there were no clinical findings related to it. Roentgenograms showed massive vertebral bone structure, especially in the pedicles. Enlargement of pedicles had caused decrease of IPD's (Fig. 7, *A* and *B*). Sagittal diameters of her lumbar spinal canal were also diminished.

Case iv. E.F., a 3 year old male, suffered from osteochondrodystrophy, basilar invagination, partial atlanto-occipital assimilation, hemivertebra at T2 and ventral hemivertebra at T10. There were no clinical findings which could be related to stenosis of the spinal canal although roentgenograms showed gross decrease of IPD's in the lower thoracic spine. At the same level, there was marked increase of mid-sagittal diameter of the spinal canal. Roentgenographic abnormalities and a plot of the patient's IPD's are shown in Figure 8, *A* and *B*.

SUMMARY

In this article refined maximum and minimum interpediculate distances are published for use in diagnosis of intraspinal tumor and detection of spinal canal stenosis. The subject of spinal canal stenosis due to decrease of interpediculate distances is virtually unexplored. Availability of minimum norms should facilitate such studies.

Concerning spinal canal stenosis due to diminished interpediculate distances, it is apparent that (a) there is such an anomaly, (b) it may, but need not be, associated with diminished mid-sagittal diameter of the spinal canal, and (c) it may be symptomatic. Illustrative cases are presented.

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PERIOSTEAL BONE GROWTH IN NORMAL INFANTS

A PRELIMINARY REPORT

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IN 1930 Gravinghoff[6,7] said, "In every periosteal reaction in childhood you can hardly doubt congenital syphilis as the diagnosis." Syphilis has now moved to the background and other conditions have come to the forefront as causes for periosteal new bone. Among these are infection, infantile cortical hyperostosis,[4] unrecognized trauma,[12] hypervitaminosis A,[2] leukemia,[13] and neuroblastoma,[9] to mention only a few.

Little thought has been given to roentgenologically visible periosteal new bone as a finding in normal infants. Malmberg[11] found it on the long bones in 54 of 114 (47 per cent) premature infants. He concluded that it was caused by rickets and could be prevented with large doses of vitamin D. He did say, "Proliferation of the periosteum seems to occur in certain non-treated infants without subsequent appearance of rachitic signs." Glaser[5] reported a "double contour" of the long bones in 78 of 100 premature infants and in 46 of 100 full term infants. He considered it a normal roentgen appearance caused by projection of the roentgen rays in a certain direction relative to the bone, its crests and the film, and supported his contention by roentgenograms taken of the same bone in different positions relative to the direction of the rays.

Hancox *et al.*[8] also described the "double contour" of the long bones in 20 of 73 healthy infants and concluded that it is due to a local exaggeration of the normal appositional growth of the bone. Caffey[3] mentions "cortical thickening of prematurity" as occurring in more than half of all nonsyphilitic premature infants and emphasizes that they show no evidence of other disease. He suggests mild trauma incidental to ordinary care and medical examination as the cause.

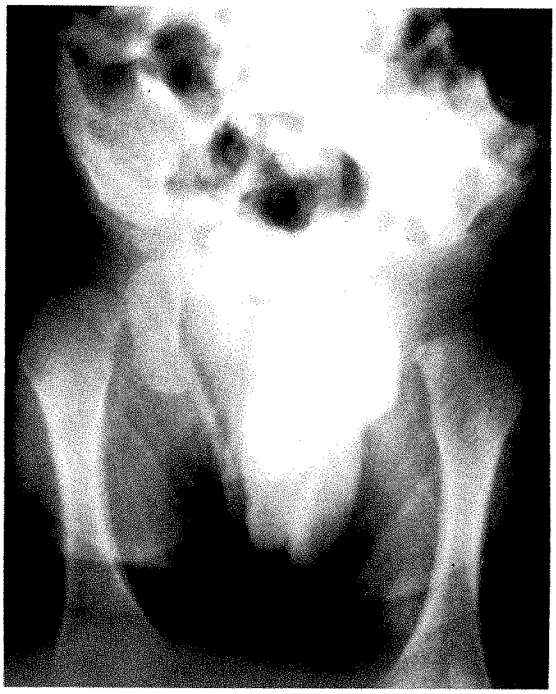

FIG. 1. Six week old white male who was admitted to the hospital for repair of an inguinal hernia. A mass was palpable in the right lower quadrant which was shown on the intravenous pyelogram to be due to crossed fused ectopy of the kidneys. Periosteal new bone was incidentally noted on each femur. There were no symptoms or abnormal physical findings relative to the femora. Periosteal new bone was also found on each tibia and humerus.

The incidental observation of periosteal new bone on the long bones is common in pediatric radiology; for example, the humeri on chest examinations and the femora on abdominal roentgenograms (Fig. 1). The purpose of the author is to report the results of a roentgenologic study of the long bones in normal full term and premature infants to determine the incidence, roentgen char-

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acteristics, pathogenesis and significance of this type of periosteal new bone.

MATERIAL

The long bones were examined in 2 groups of patients. Group I includes 335 full term infants under 6 months of age who had no symptoms referable to the skeleton. One hundred and sixteen of these were well babies and the long bones were examined primarily for this study. The remaining 219 patients were referred to the radiology department for chest examinations and all were well babies prior to the onset of acute upper respiratory symptoms. Eighty-eight of these infants were initially examined during the first month of life and the remainder after this age. At least one serial examination at a 1 month interval was performed on 135 of the total group.

Group II consists of 75 premature infants who were developing satisfactorily and had no known systemic disease or skeletal symptoms. Sixty-six of them had at least one serial examination at a 1 month interval. They were all examined initially during the first month of life.

INCIDENCE

Periosteal new bone was present in 115 of the 335 (35 per cent) full term infants. The femora were most often involved followed in frequency by the humeri, tibiae, ulnae and radii. The involvement of individual bones is listed in Table I.

The new bone is invariably bilateral. No instance of unilateral involvement of any of the bones is recorded, although it is occasionally more prominent on one side than the other.

Table I

INCIDENCE OF PERIOSTEAL NEW BONE IN 335 FULL TERM INFANTS

Bones	Incidence	
	Number	Per Cent
Femora	108	32
Humeri	103	30
Tibiae	98	29
Ulnae	68	20
Radii	34	10

Table II

INCIDENCE OF PERIOSTEAL NEW BONE IN 75 PREMATURE INFANTS

Bones	Incidence	
	Number	Per Cent
Femora	23	31
Humeri	23	31
Tibiae	22	30
Ulnae	11	15
Radii	8	11

The femora, humeri and tibiae are affected almost equally. On numerous occasions, new bone may be limited to one pair of bones but becomes evident on the uninvolved ones in a subsequent examination. The discrepancy between the over-all incidence of 35 per cent and that recorded for the femora, humeri and tibiae individually may be explained by the absence of a follow-up examination in the few infants who had only one pair of bones involved. It appears to be a valid rule that the femora, humeri and tibiae will all be involved if one of them is. The same rule does not hold for the radii, ulnae and fibulae and there is no explanation suggested for the decreased involvement of these bones.

The incidence of new bone is the same in the premature infants as in the full term ones, being present in 26 of the total group of 75 (34 per cent). Individual bone involvement for this group is listed in Table II. The bilateral and equal affection of the femora, humeri and tibiae is observed also in the premature infants.

Age is a factor which definitely influences the incidence. Periosteal new bone was not seen before the age of 1 month in the full term infants and occurred only once in the premature infants prior to this age. The incidence rises to 47 per cent in the full term infants and to 39 per cent in the premature ones if those examined before the age of 1

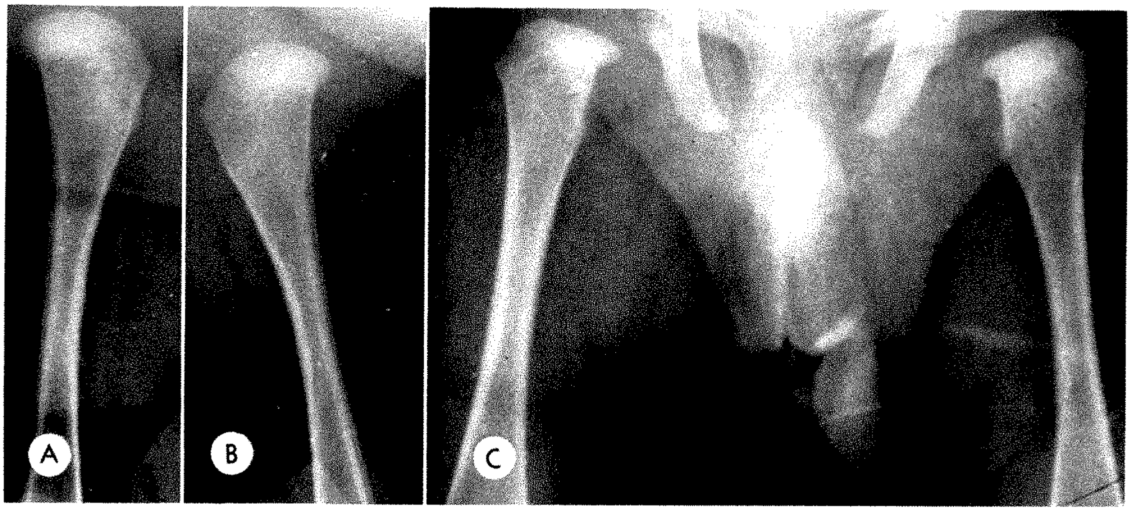

Fig. 2. Six week old full term male referred from the well baby clinic. (*A* and *B*) Lateral roentgenograms of each femur show a thin, smooth layer of new bone on the anterior aspect. (*C*) Anteroposterior roentgenogram of the femora does not show the new bone.

month without follow-up study are excluded.

No difference in incidence was detected in the 116 babies from the well baby clinic and the 219 who were referred for chest examinations because of upper respiratory symptoms.

ROENTGEN CHARACTERISTICS

The roentgen appearance of the periosteal new bone in this group of full term and premature infants depends upon the age at the time of the examination. It is a delicate, smooth layer in infants between the ages of 1 to 2 months (Fig. 1; and 2, *A*, *B* and *C*). The new bone is not always concentric in its distribution and is present on only one aspect of the bones in some (Fig. 2, *A*, *B* and *C*; and 3, *A*, *B* and *C*). There is no consistency as to the site of involvement except in the tibia were the new bone is invariably on the medial aspect. It is always on the diaphysis and usually extends onto the metaphysis to a variable extent, but it has not been observed to reach the end of the metaphysis (Fig. 1; 2, *A*, *B* and *C*; and 3, *A*, *B* and *C*).

This type of periosteal new bone becomes quite thick in some areas. In spite of the thickness it does not form multiple layers. Instead, there is one amorphous, homogenous layer which maintains its smoothness (Fig. 3, *A*, *B* and *C*). The thickness attained is variable in the bones of the same infant as well as in the individual bones of different infants.

Those infants who had sequential examination at approximately 1 month intervals show that the initial fine layer of new bone thickens to a variable degree, matures, and becomes incorporated into the cortex (Fig. 4, *A*-*F*). There is a thin translucent zone which separates the layer of new bone from the cortex. This forms a roentgen image of two parallel dense lines separated by the translucent zone, the inner line being the cortex and the outer line the layer of new bone. The cortex is resorbed from within and the layer of new bone becomes the new cortex. The bones actually have a double cortex before the old cortex is completely resorbed. It seems without question that this double cortex is the double contour referred to by Glaser[5] and Hancox *et al.*[8]

No specific search was made for the double cortex as a part of this study, but it has been observed incidentally in infants between 1 and 2 years of age. Glaser[5] found its peak incidence to be between 3 and 6 months of age, which would be expected

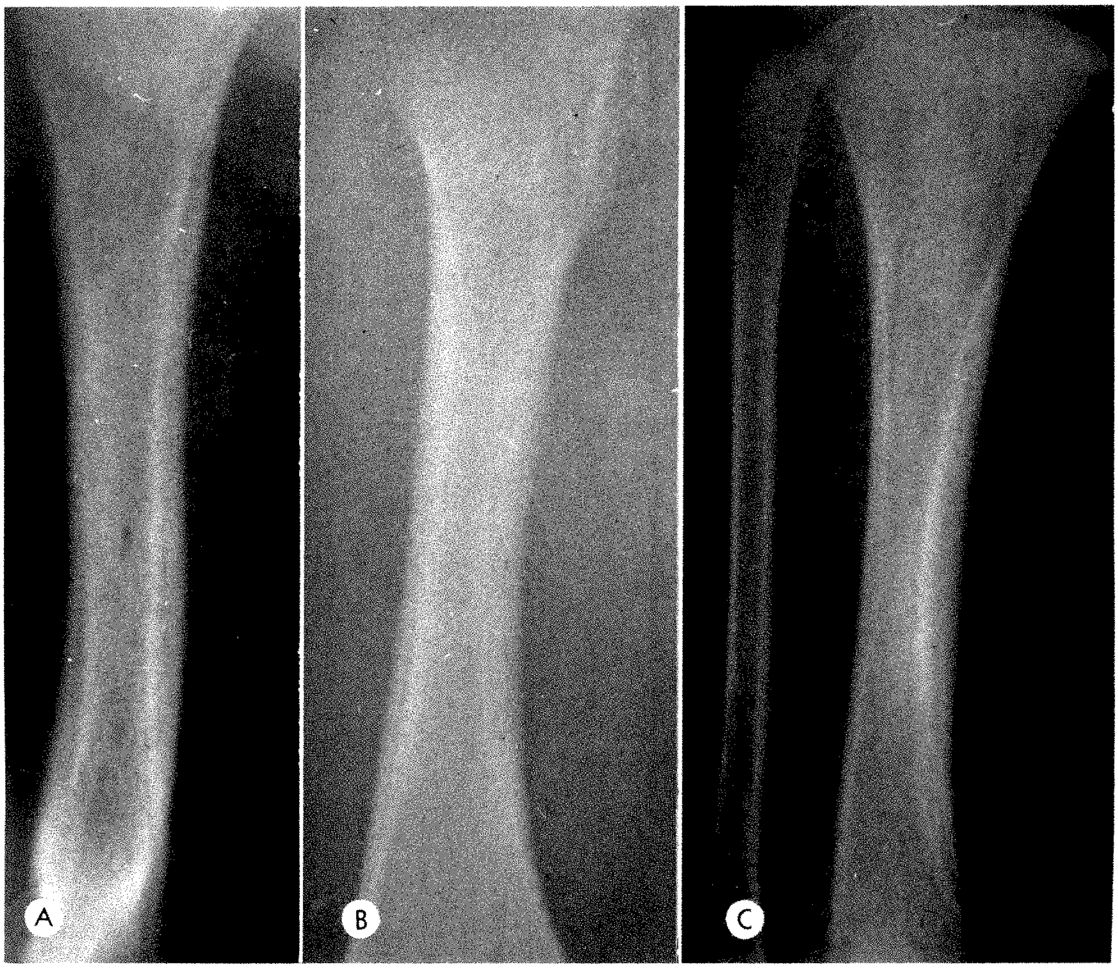

FIG. 3. Two month old full term male who was well until acute respiratory symptoms appeared 2 days prior to examination. (*A*) The right humerus has new bone on each margin. (*B*) New bone is present on the lateral margin of the right femur. (*C*) A thick layer of new bone is present on the medial margin of the right tibia. Observe that the new bone involves primarily the diaphysis but extends onto the metaphysis without reaching its end. (The opposite humerus, femur and tibia also had similarly appearing new bone.)

from the information obtained from our study. The presence of the double cortex in the older infants indicates that the time required for complete resorption of the old cortex is inconstant (Fig. 5, *A–D*).

Glaser[5] also found the double cortex to be more common on the ulna and radius than on the femur, humerus or tibia. This does not conform to the distribution of the periosteal new bone found in our infants, and suggests that the double cortex may be formed by new bone which is not roentgenologically visible in the earlier stages. This concept is supported by the fact that many times new bone will be seen on only one aspect of a bone, but in subsequent roentgenograms the cortex has increased equally in width on the side where there was no new bone. Perhaps the periosteum has formed new bone concentrically, but it is roentgenologically detectable in just one area (Fig. 5, *A–D*; and 6, *A* and *B*).

These sequential changes through which the periosteal new bone passes account for the different roentgen appearance at the various ages. A thin, delicate layer of new bone has not been observed after the age of 3 months. Instead, the double cortex image

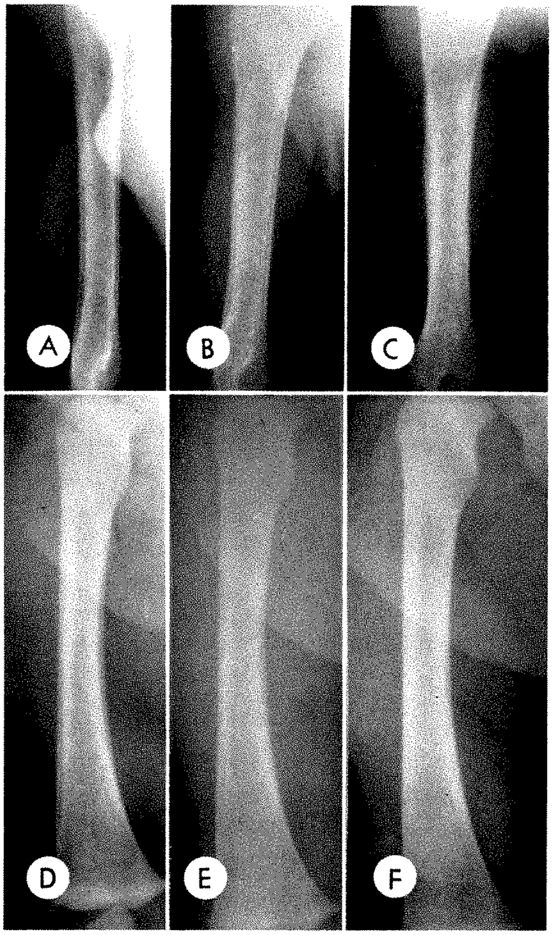

FIG. 4. Right humerus and femur of a full term, well female infant. (*A*) Right humerus at the age of 5 weeks. New bone is well developed. (*B*) The bone has thickened, matured and formed a double cortex at the age of 9 weeks. (*C*) The old cortex has been almost completely resorbed at the age of 13 weeks and the humeral diaphysis has increased in width by the thickness of the new bone. (*D*, *E* and *F*) The right femur at the same ages as *A*, *B* and *C*. The new bone on the femur has shown the same changes as seen in the humerus.

is found and is evidence that new bone was present earlier (Fig. 6, *A* and *B*; and 7, *A* and *B*).

A significant roentgen feature is that the bone structure underlying the periosteal new bone is perfectly normal in appearance (Fig. 1 through 7).

ETIOLOGY

The proclivity and ease with which the infant's periosteum forms new bone is well documented.[3,14] It has been related to the facts that the periosteum is loosely bound to the underlying cortex because of the sparsity and shortness of Sharpey's fibers, and that the cambium layer of young periosteum has increased osteogenetic activity (Fig. 8).

Caffey has suggested that mild trauma, incidental to ordinary care and medical examination, is enough to provoke the loosely attached and osteogenetically active periosteum to form new bone. This hypothesis was tested in 10 normal infants between the ages of 1 and 2 months. Roent-

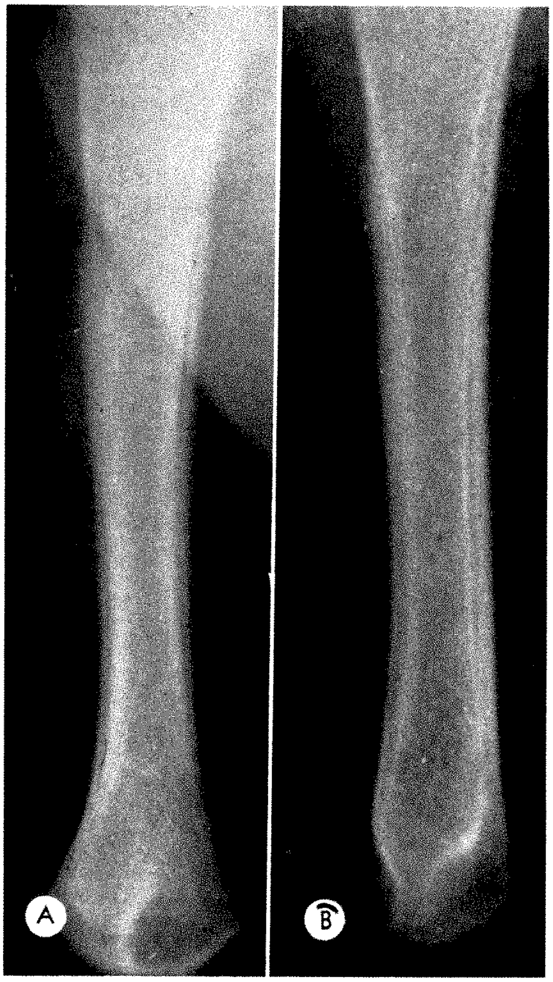

FIG. 5. (*A* and *B*) Progressive changes of the new bone on the humerus of a full term, well male infant are seen at ages 8 and 13 weeks, respectively.

Fig. 5. (*C* and *D*) Similar changes on the right femur at the same ages as *A* and *B*. The old cortex of the femur has been resorbed (*D*) but that of the humerus is still present (*B*), illustrating the difference in rate at which this process proceeds in the various bones of the same patient. The cortex of the medial and lateral margins of the femur is visibly thicker in *D* than it is in *C*. However, new bone was visible only on the medial margin.

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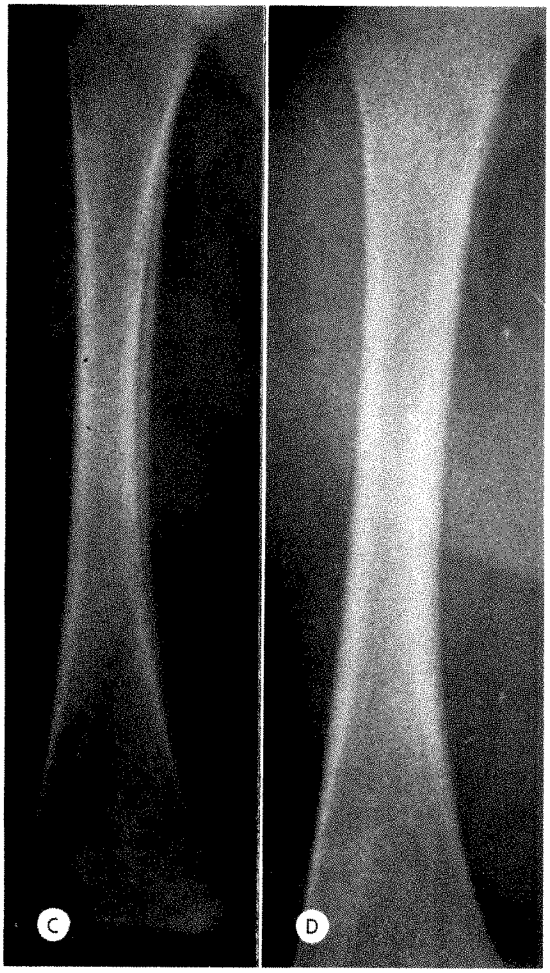

genograms of each humerus were taken as a baseline and 3 of the 10 showed periosteal new bone bilaterally, typical of that seen in the other infants in this study. One humerus of all 10 infants was massaged and the opposite one was not. The roentgenographic examination was repeated 2 and 4 weeks after the massage. No difference in the appearance of the periosteal new bone was found in the 3 infants who had demonstrated it bilaterally in the baseline roentgenograms. Each showed identical progression of the new bone as observed in the other infants of the study. One of the 7 infants without new bone demonstration in the baseline roentgenograms demonstrated it in the subsequent roentgenograms, but it was identical in appearance bilaterally.

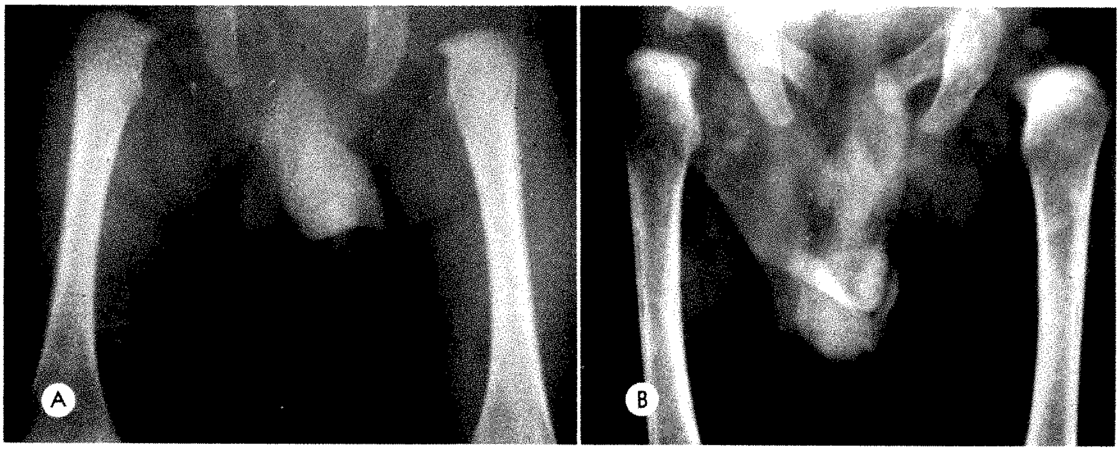

Fig. 6. Full term well, male infant. (*A*) Femora at the age of 6 weeks showing new bone on the lateral margins. (*B*) Femora at the age of 6 months. A well formed double cortex is present on each femur laterally. The cortex medially is thicker with a faint double cortex, even though new bone is not seen at this area in *A*. The part played by the layer of periosteal new bone in the pathogenesis of the double cortex seems clear.

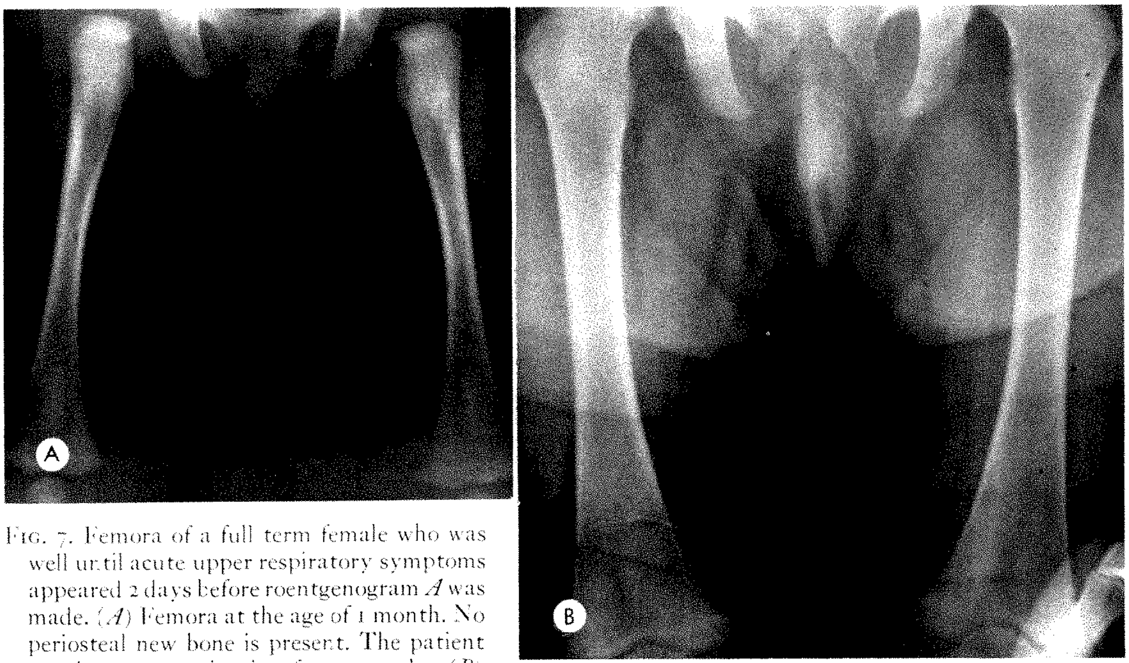

FIG. 7. Femora of a full term female who was well until acute upper respiratory symptoms appeared 2 days before roentgenogram *A* was made. (*A*) Femora at the age of 1 month. No periosteal new bone is present. The patient was lost to examination for 7 months. (*B*) Femora at the age of 8 months. A double cortex is present bilaterally. The pathogenesis of the double cortex indicates that periosteal new bone appeared within 1 to 2 months after *A* was made and produced the double cortex seen at the age of 8 months.

The humeri are sometimes held quite tightly by technicians to immobilize patients for chest examinations. It seems possible that enough trauma could be produced to initiate periosteal new bone formation, if indeed, trauma is the cause. However, 1 month interval roentgenograms of the humeri of 25 patients having such chest examinations failed to reveal any evidence to support trauma, incident to the examination, as a cause for periosteal new bone.

It is readily admitted that these crude and simple observations do not exclude a possible role by trauma, but the incidence of 35 per cent in normal infants makes it seem unlikely. It does not seem possible that ordinary care and medical examination would produce trauma sufficient to stimulate periosteal new bone in one-third of the healthy infants.

Other factors which are difficult to reconcile with a traumatic etiology are the symmetry and multiplicity with which the bones are affected, the evenness and smoothness of the new bone, the failure of the new bone to reach the end of the metaphysis, and the occurrence of the new bone after the age of 1 month. It would seem that birth trauma would be a sufficient stimulus, but if so, the new bone should appear by the age of 2 weeks and would not be expected to be completely symmetric.

These same factors support the theory that the periosteal new bone is a manifestation of normal growth. The long bone diaphyses of the fetus are formed by intramembranous ossification.[1,10] After birth, the periosteum retains this intramembranous type of bone formation in which the cambium cells have the ability to form osteoid without a preformed cartilage scaffolding. It is by this process that the diaphyses of the growing long bones increase in width. At the same time, resorption is occurring on the inside of the cortex, but because it is less than the amount of bone formed, thickening of the cortex also occurs.

It seems most logical that the periosteal new bone found in these infants is simply a roentgen manifestation of normal periosteal

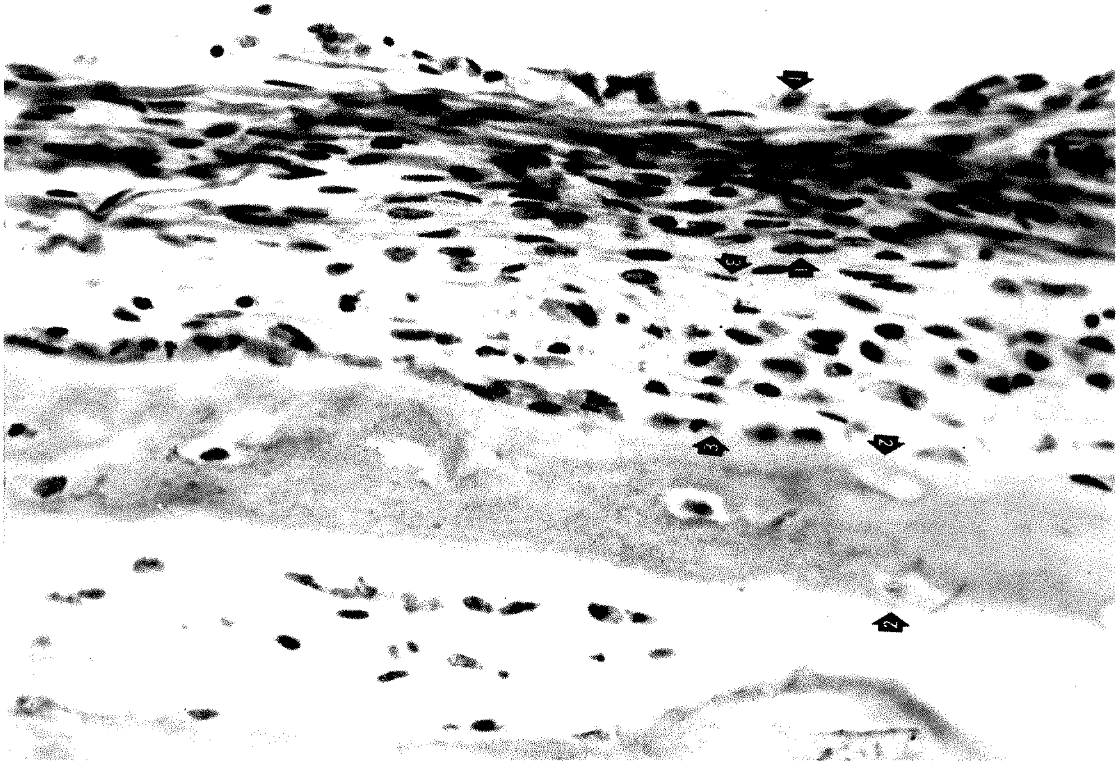

Fig. 8. Section taken through the periosteum of a fetal bone. Periosteum is composed of two layers. The outer (arrows—1) is the fibrous layer and is composed of spindled collagenoblasts. Between this layer and the cortex (arrows—2) is the cambium (arrows—3) which is the bone forming layer. It is thickened because of its increased osteogenetic activity, whereas in the adult it does not exist as a layer unless revived by injury or a disease process. (Reproduced by permission from Aegerter, E. and Kirkpatrick, J. A., Jr., Orthopedic Diseases, Second edition, 1963. W. B. Saunders Company, Philadelphia.)

bone growth in the period of greatest activity. No explanation is certain as to why all infants do not show it. Perhaps there is a normal differential in the rate of bone formation by the young periosteum and only those showing the most rapid growth are roentgenologically evident. It is distinctly possible that the method of examination as employed in our study is deficient. This possibility is supported by the facts that the incidence is higher if only those infants older than 1 month are considered and that the double cortex is found in a higher percentage.[5,8]

None of the patients in our study had biopsy. Hancox *et al.*[8] report histologic studies of 2 infants exhibiting the double cortex. In each instance, the bone tissue in the region of the double cortex appeared normal histologically. The trabeculae were distributed in such a way as to suggest that there might be two outer boundaries to the shaft of the bone, and resembled the pattern encountered where bone deposition is proceeding rapidly, such as in fracture callus.

One thing is certain, the infants in this study had no symptomatic, physical, or roentgenologic evidence of bone or systemic disease.

SIGNIFICANCE

The periosteal new bone observed in the infants of this study is of no significance to the patient himself because it appears to be a manifestation of normal periosteal bone growth and acts in a perfectly benign manner.

It has considerable significance to the physician who can be presented with a difficult problem in differential diagnosis. The long bones of infants between the ages of 1 and 6 months of age commonly are examined for evidence of trauma and certain disease processes. It is an understatement to say that this type of periosteal new bone must not be interpreted as being due to disease.

The features which assist in its recognition are the bilaterality and multiplicity of involvement, absence of abnormality in the underlying bone, and the sequential conversion of it into a new cortex.

The list of conditions to be considered in the differential diagnosis is a long one. Included are syphilis, trauma, infection, infantile cortical hyperostosis, rickets, scurvy and hypervitaminosis A.

Syphilis almost always causes underlying change of the cortex or spongiosa in the form of areas of destruction or bands of diminished density. In such instances the accompanying periosteal reaction is wavy, irregularly thickened and laminated. A single layer of periosteal thickening on multiple bones without underlying destruction has been described as a finding of syphilitic periostitis. This would pose a most difficult problem. The serology might be helpful but one should not necessarily conclude that a positive result indicates syphilitic periostitis. Sequential study may be the best way to distinguish the two types of periosteal response since syphilitic periostitis usually lasts for many months.

Traumatic periosteal new bone may be bilateral and multiple, but there is usually evidence of fracture or hematoma. The new bone is uneven and extends to the end of the metaphysis in some areas.

Infection as a cause for periosteal new bone without evidence of metaphyseal destruction is unlikely. The new bone will not affect multiple bones symmetrically and the resolution will be prolonged and irregular.

The clinical picture of infantile cortical hyperostosis is usually characteristic. The mandibles, clavicles, and ulnae are involved most frequently. The periosteal bone extends to the end of the metaphysis, may be localized, and is bulky, uneven and laminated.

Rickets and scurvy can be excluded by the absence of the characteristic metaphyseal changes of the two diseases. The new bone of scurvy is thickened and wavy, and extends to the end of the metaphysis.

Hypervitaminosis A causes loss of appetite, itching and fretfulness in its early stages. Later, soft tissue lumps with underlying cortical thickening appear on several bones, but the ulnae and metatarsals are consistently affected. The periosteal bone shows localized irregularity and thickening and is not symmetric and multiple in its location.

DISCUSSION

The findings reported in this study are preliminary and do not justify far-reaching conclusions. A need exists for a more detailed sequential study of infants between 1 and 3 months of age and a correlation of the roentgen findings with biopsy studies. The problems involved in obtaining biopsies on asymptomatic, healthy infants are obvious. However, it is from such studies that a better understanding of the problem will result.

Nevertheless, the current findings seem valid enough to substantiate the opinion that periosteal reaction on the long bones of infants, between the ages of 1 and 6 months, is not rare and necessarily abnormal. On the contrary, it is common and is not an indication for treatment unless a definite relationship with a specific disease is proven.

The presence of periosteal proliferation in a patient with some known bone or systemic disease may be an incidental finding and is not necessarily part of the disease. It seems possible that in the past the roentgen manifestations of normal periosteal bone growth may have been wrongly described as components of some diseases.

SUMMARY

1. Preliminary results of a roentgenologic study of the long bones in 335 full term and 75 premature infants are presented.

2. Periosteal new bone occurred in 35 per cent of both groups in the absence of other signs of bone disease.

3. The roentgen features and differential diagnosis are discussed.

4. The new periosteal bone passes through an orderly, benign conversion into a new cortex for the involved bones.

5. It seems most likely that the new bone is a roentgen manifestation of rapid periosteal bone growth.

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ANGIOCARDIOGRAPHIC OBSERVATIONS IN THE POST-RUBELLA SYNDROME*

PRELIMINARY REPORT

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IT IS well recognized that the young infant with congenital heart disease following maternal rubella has a tendency for certain anomalies of the cardiovascular system.[1-4] These anomalies are usually represented by (1) the isolated patent ductus arteriosus, (2) patent ductus arteriosus occurring in association with stenotic lesions of the pulmonary valve and/or pulmonary arteries, and (3) stenoses of the central or peripheral pulmonary arteries without additional cardiac anomalies.

During the last 6 months, we have observed 9 infants in which it had been definitely established that the mother contacted German measles during the first trimester of pregnancy. As each patient exhibited clinical and roentgenographic findings of a left-to-right shunt, retrograde aortography was performed. In addition to opacification of a patent ductus arteriosus in each case, the main pulmonary artery and pulmonary valve cusps were visualized as well.

As our experience developed, it became apparent that in addition to a patent ductus arteriosus, there was a distinct tendency for a relatively constant anomaly of the main pulmonary artery and valve to occur in 8 of the 9 cases studied. In the ninth case (the only case examined at necropsy), the main pulmonary artery and valve were normally formed. As a result, this case will not be included in the following discussion.

The anomalies observed in the other 8 cases were as follows: (1) a foreshortened main pulmonary artery, (2) a high posteriorly placed vertically tilted pulmonary valve, (3) pulmonary valvular regurgitation, and (4) thickened pulmonary valves. In addition, 2 of these 8 cases demonstrated localized ectasia of the left subclavian artery.

ANGIOGRAPHIC DEFINITION OF THE ANOMALIES (8 CASES)

1. *Pulmonary Trunk.* In each case studied, the pulmonary trunk was foreshortened. In other words, the distance between the pulmonary valve cusps and the main division into the right and left pulmonary artery branches was diminished (Fig. 1, *a* and *b*). The degree of foreshortening varied from moderate to severe. In 5 cases, the pulmonary valve was projected so posteriorly on the lateral view that it overlay the ascending aorta. In order to appreciate the aforementioned findings of the main pulmonary artery and valve, the pulmonary valve and artery in an infant with patent ductus arteriosus without rubella infection are shown in Figure 2*a*.

2. *Pulmonary Valve.* In contrast to the normal, the plane of the pulmonary valve as viewed in the lateral projection appeared to be in a vertical or near-vertical position (Fig. 1*a*). Owing to this vertical position, the contrast filled sinuses could be seen *en face* during ventricular systole.

3. *Pulmonary Insufficiency.* In 6 of the 8 cases, there was evidence of incompetence of the pulmonary valve. This was shown by regurgitation of a considerable amount of

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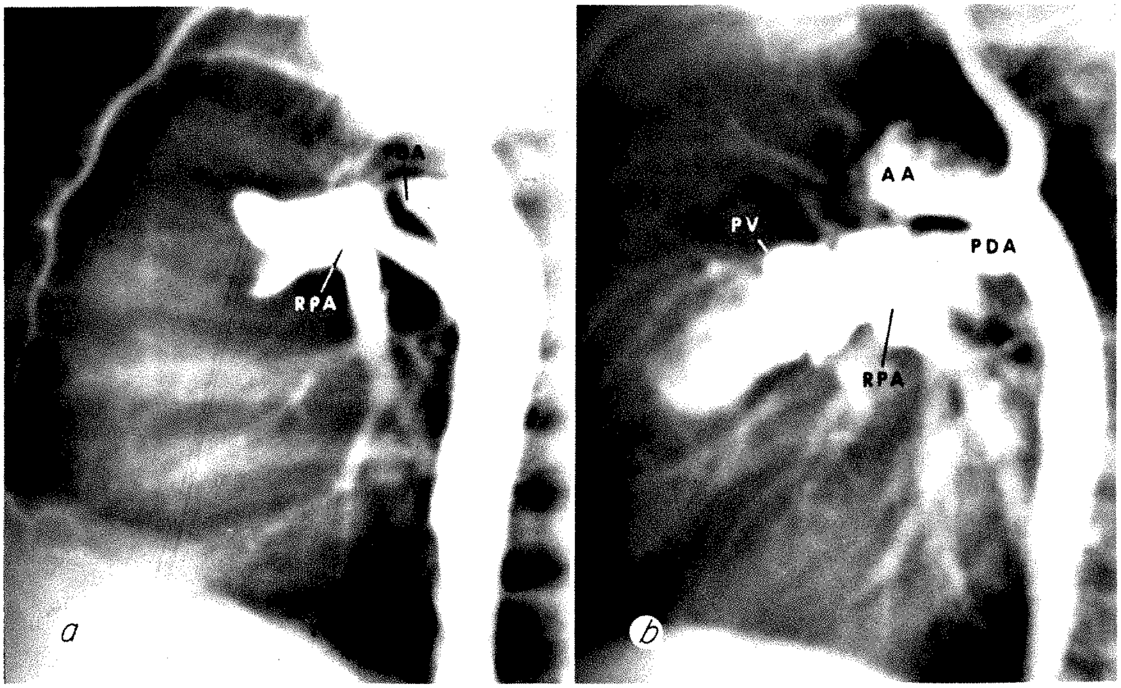

Fig. 1. Retrograde aortograms of 2 infants with the post-rubella syndrome and patent ductus arteriosus (PDA). (*a*) Lateral view. The main pulmonary artery is foreshortened. The distance between the vertically tilted pulmonary valve and origin of the right pulmonary artery (RPA; seen on end) is diminished. (*b*) Lateral view. The main pulmonary artery is foreshortened as in *a*. The pulmonary valve (PV) is thickened and has allowed a considerable degree of pulmonary insufficiency. AA = aortic arch.

contrast material through the pulmonary valve during diastole (Fig. 1*b*).

4. *Thickening of the Pulmonary Valve.* In 2 cases, the leaflets of the pulmonary valve were distinctly thickened (Fig. 1*b*). In both cases, pulmonary insufficiency was present.

5. *Localized Dilatation of Systemic Arteries.* There was localized dilatation of the left subclavian artery in 2 cases (Fig. 2*a*).

DISCUSSION

Recent emphasis in the literature has focused attention on the association of the post-rubella syndrome with central and peripheral pulmonary artery stenosis. We are presenting this group of anomalies as representing possibly a sequela of the post-rubella syndrome, too, since it had been established in each case that the mother had contacted German measles during the first trimester of pregnancy. Moreover, these anomalies are usually not observed in the "non-rubella" patent ductus arteriosus.

What precise mechanism causes this specific group of anomalies is unknown. There does, however, seem to be a definite predilection of the measles virus for affecting the development of the pulmonary valve and vascular system. Localized ectasia of the left subclavian artery (2 cases) suggests that the measles virus may affect the systemic arteries as well.

The cause of pulmonary insufficiency is very likely an abnormal pulmonary valve, which appeared to be distinctly thickened in 2 cases. The cause for pulmonary insufficiency in the other 4 cases, however, is less obvious. To date, no postmortem confirmation of these 4 cases has been obtained. Dilatation of the pulmonary artery causing mechanical distortion of the pulmonary valve cusps is another possibility. This, however, seems unlikely since in contrast to cases of patent ductus arteri-

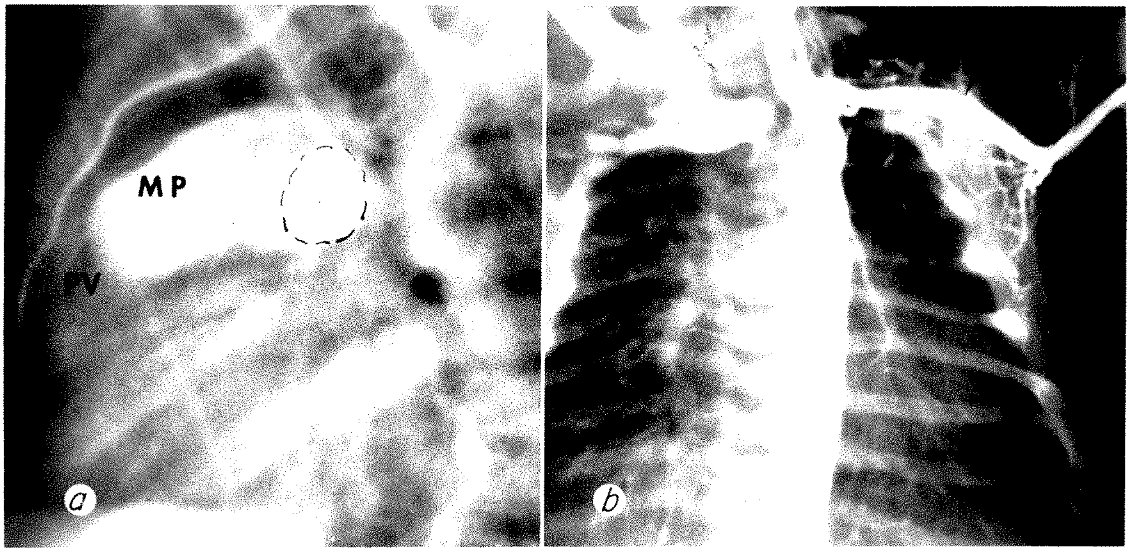

Fig. 2. (*a*) Retrograde aortogram in infant with patent ductus arteriosus of the non-rubella variety. The main pulmonary artery (MP) is dilated, but shows considerable more length between the pulmonary valve (PV) and right pulmonary artery (circled) than that observed in Figure 1, *a* and *b*. (*b*) Anterior retrograde aortogram showing localized ectasia (arrow) of the left subclavian artery. The injection had been completed at time of this exposure.

osus of the non-rubella type, the pulmonary trunk was not significantly dilated. When pulmonary insufficiency occurs in the "non-rubella ductus," it is usually due to dilatation of the pulmonary trunk secondary to pulmonary hypertension.

Lastly, the described findings seem to be specific for post-rubella patent ductus arteriosus, but apparently not uniformly present. This is indicated by our ninth patient who exhibited no pulmonary insufficiency. In addition, the anatomy of the pulmonary artery was normal at time of necropsy.

SUMMARY

Angiocardiographic findings in 9 infants with patent ductus arteriosus following maternal rubella are described. In 8 patients, certain anomalies of the pulmonary valve and main pulmonary artery were present which represent possibly a specific sign of post-rubella infection. These anomalies are: (1) a foreshortened main pulmonary artery, (2) a high, posteriorly placed, vertically tilted, pulmonary valve, (3) pulmonary valvular regurgitation, and (4) thickened pulmonary valves. In addition, 2 cases showed localized ectasia of the left subclavian artery.

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AORTIC RING CAUSED BY DOUBLE AORTIC ARCH IN AN ADULT*

ANGIOCARDIOGRAPHIC REPORT OF A CASE

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A DOUBLE aortic arch compressing the trachea and esophagus is exceedingly rare in adults. Gross,[2,3] in 1955, when summarizing his experience with this lesion in 26 cases, reported that their ages varied from 1 month to 3 years. Early in life, the patient herein reported, the daughter of a physician, had stridor which had been attributed to a congenitally small trachea; this was followed by chronic dyspnea on exertion. Not until her nineteenth year was the definitive diagnosis of double aortic arch compressing the trachea and esophagus established by angiocardiography; cure by ligation and division of the smaller arch followed.

REPORT OF A CASE

A college student, referred by Dr. Irving J. Kane, complained that all her life she had been dyspneic on exertion, compelling her to curtail physical activities. Soon after going to a college which had an unusually hilly campus, the dyspnea increased. The mother reported that while a stridor had been present at birth, it had disappeared. The only illness that the patient had had was pneumonia at the age of 6 months, and recovery from this had been uncomplicated. The patient denied dysphagia, weight loss, or respiratory discomfort. Physical examination disclosed a well developed and well nourished Caucasian girl in no discomfort who weighed 115 pounds and measured 64 inches in height. A faint stridor was audible during ordinary conversation while the history was taken. However, it could not be elicited during auscultation. The lungs were clear and there were no rales. The heart was not enlarged and there were no murmurs; the rhythm was regular and the pulse rate was 72 per minute. The blood pressure was equal in both arms and measured 110/60 mm. Hg. Studies of the urine, blood, pulmonary function, and electrocardiogram were within the limits of normal. The conventional frontal roentgenogram of the chest showed clear lung fields and a normal cardiac silhouette (Fig. 1*A*). The aortic shadow did not appear abnormal, but the trachea was somewhat narrowed. The frontal and lateral esophagraphic studies showed deformity of both sides of the esophagus in the region of the aortic arch, the right more than the left (Fig. 1, *B* and *C*). Laminagraphic study of the esophagus disclosed that the right-sided filling defect was larger than the left (Fig. 2). Serial angiocardiography in frontal and left anterior oblique views, on December 23, 1958, revealed bilateral aortic knobs, the right larger than the left (Fig. 3, *A* and *B*). There were two aortic arches. The larger (right) proceeded in front of the trachea and gave off normal brachiocephalic arterial branches. The smaller posterior arch joined with the anterior just beyond the origin of the left subclavian artery and became the descending thoracic aorta, proceeding along the anterior surface of the thoracic spines (Fig. 3*B*).

Surgical exploration on June 30, 1959, by Dr. J. Maxwell Chamberlain, disclosed that the right aortic arch was the larger and anterior vessel. From it arose the innominate, left common carotid and left subclavian arteries. The smaller left aortic arch commenced behind the origin of the innominate artery and passed posteriorly behind the esophagus and trachea. It joined the right aortic arch opposite the origin of the left subclavian artery. Once the anomalous posterior arch was dissected free, it was easily ligated and divided.

DISCUSSION

There is general agreement that most patients with a double aortic arch, a remnant of the reptilian phase of aortic

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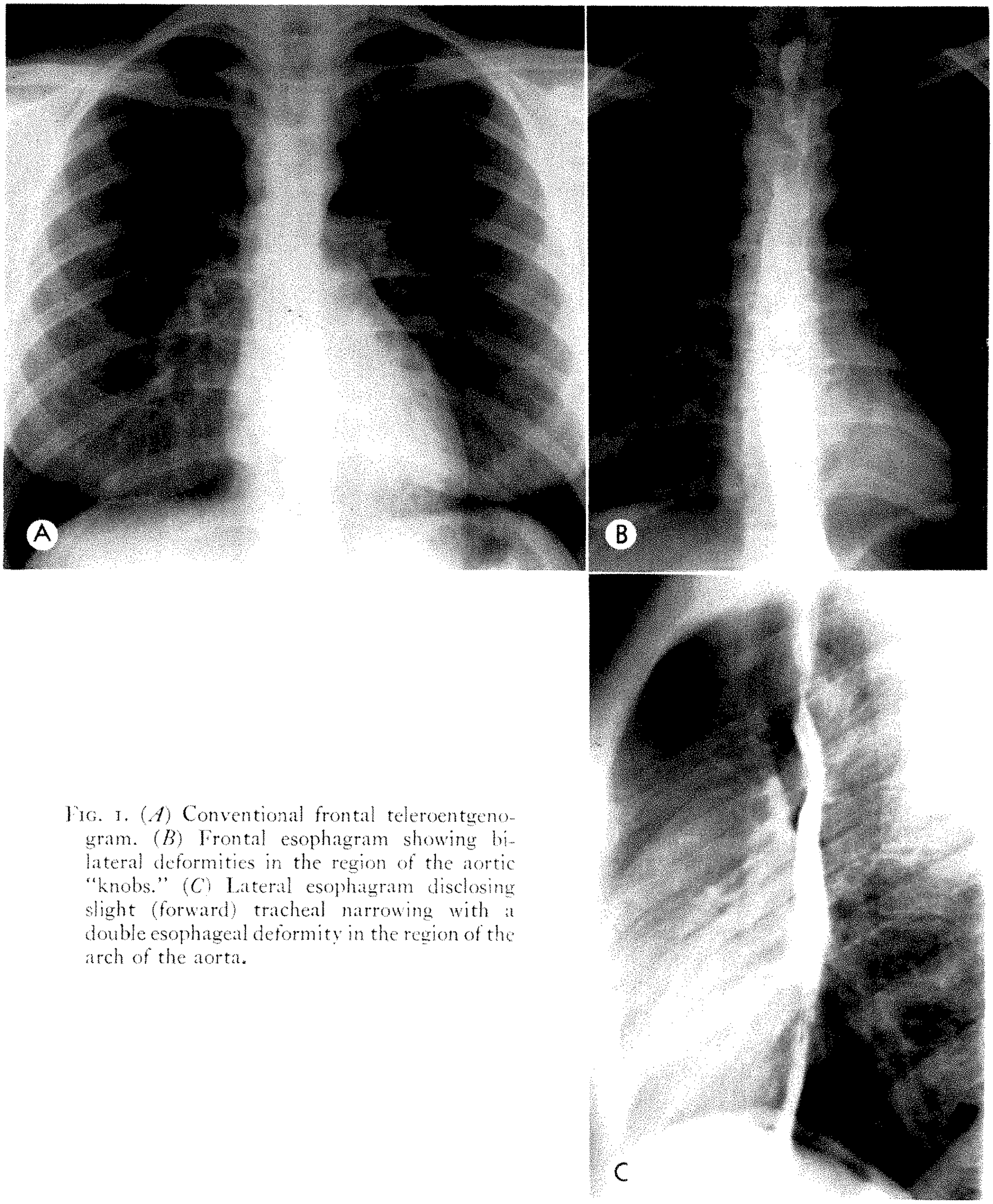
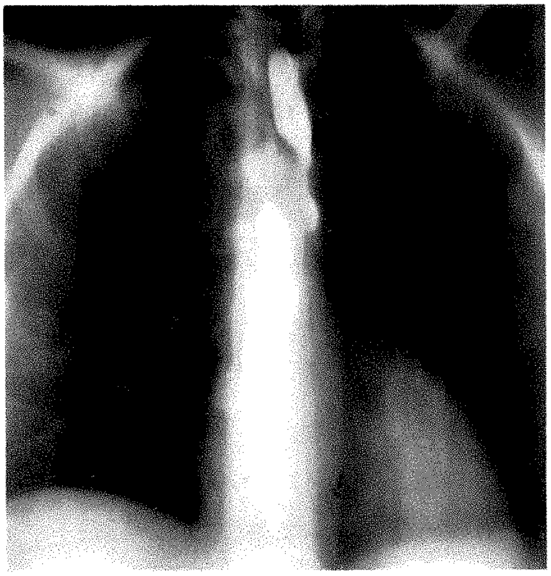


Fig. 1. (*A*) Conventional frontal teleroentgenogram. (*B*) Frontal esophagram showing bilateral deformities in the region of the aortic "knobs." (*C*) Lateral esophagram disclosing slight (forward) tracheal narrowing with a double esophageal deformity in the region of the arch of the aorta.

development, have very little room within the arterial ring, so that serious tracheal and esophageal compression results.[2,3,5] Usually, the afflicted person is an infant who presents with alarming stridor, dyspnea, cyanosis, dysphagia, and severe pulmonary infection. Prior to Gross's pioneering demonstration that the arterial ring was amenable to surgical relief,[2,3] survival beyond the seventh month of life was rare.[1]

That not all patients with a double aortic arch have severely constricting rings and are desperately ill is illustrated by the case reported above. Even though signs of

FIG. 2. Frontal laminagram of the esophagus showing the bilateral deformities in the region of the aortic knobs to advantage. Note that the right esophageal deformity is the larger.



tracheal compression had been present since birth, the patient was able to get along for many years without serious discomfort by restricting her activities. There is even a report of a 67 year old male cadaver with a classical double aortic arch which formed a vascular ring that hardly compromised the trachea and esophagus.[4]

Despite the fact that the patient herein reported had stridor, albeit faint, dyspnea, and roentgen signs of tracheal and esophageal compression (Fig. 1, *A*, *B* and *C*; and 2), she was reluctant to accept surgical exploration of the mediastinum. It was for this reason that angiocardiography was recommended. Once this was accomplished (Fig. 3, *A* and *B*), assurance could then be given that the lesion was amenable to surgical ligation and cure; and so it proved.

SUMMARY AND CONCLUSIONS

Herein is reported the rare case of an

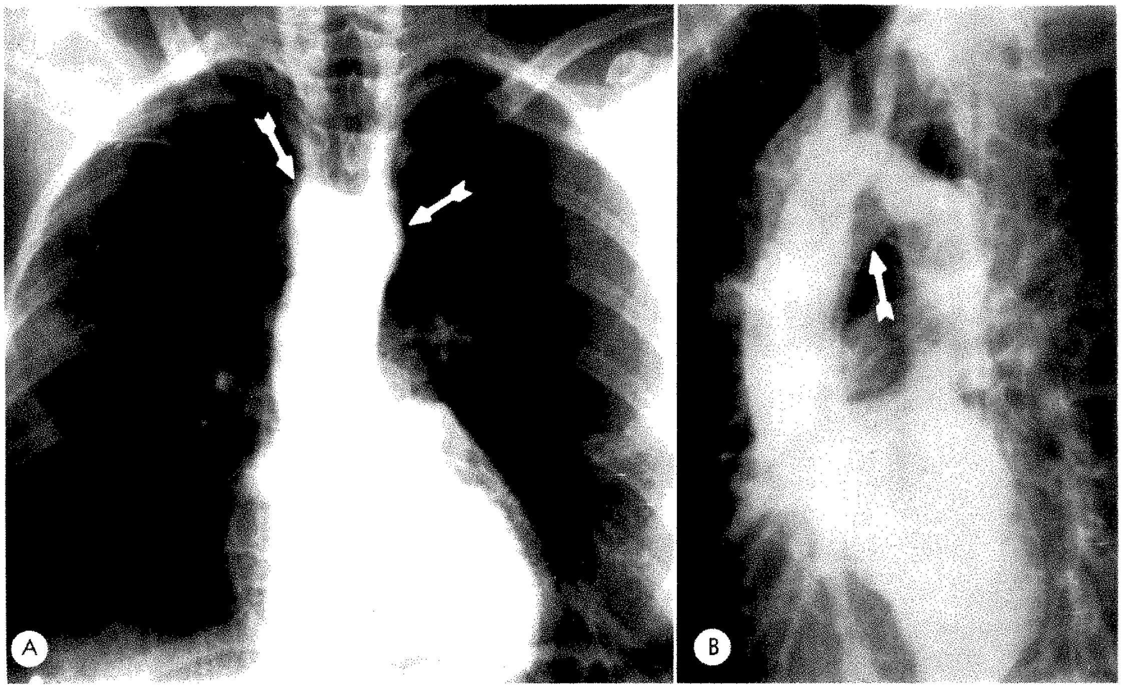


FIG. 3. (*A*) Frontal angiocardiogram showing double aortic "knobs" (arrows). (*B*) Left anterior oblique angiocardiogram revealing the larger (anterior) aortic arch with the branchiocephalic arteries. The smaller (posterior) aortic arch (arrow) begins opposite the origin of the innominate artery and joins the anterior arch in the region of the left subclavian artery to form a normal descending aorta.

adult with a double aortic arch constricting the trachea and esophagus for 19 years. Conventional roentgenographic studies disclosed the classic tracheal and esophageal deformities of a vascular ring. Although suffering from mild stridor and increasing dyspnea, and even though diagnosis of double aortic arch was clinically suspected, the patient was reluctant to undergo mediastinal exploration until the double aortic arch was visualized. Once this was accomplished and the diagnosis of a double aortic arch confirmed by angiocardiographic studies, dissection, ligation and division of the smaller posterior arch were curative.

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FURTHER OBSERVATIONS ON THE VERTICAL FISSURE LINE*

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DAVIS[2] in 1960 first called attention to a roentgenologic finding not previously described in the literature: the occasional appearance of a vertical line in the base of the lungs of infants and children. Davis observed this finding in 29 cases, and considered the line to be the main interlobar fissure appearing for some reason in the sagittal projection of the chest. A further report was published by Webber and O'Loughlin,[6] who observed the finding in 26 cases. Since Davis' publication, we have collected 37 similar cases. These are discussed, together with an attempt to elucidate the anatomic basis of this recently recognized roentgenologic phenomenon and possibly its diagnostic value.

MORPHOLOGIC DESCRIPTION

A fine linear shadow, seldom wider than 1 mm., commencing in the costophrenic angle or somewhat medially from it, runs upwards slightly inclined toward the hilus, and terminates before reaching its level. The laterally situated lines are straight; those beginning more medially are slightly curved, with a lateral convexity. The lines may be found on either side, more often on the right, and sometimes they are bilateral.

MATERIAL*

Out of our 37 cases, 26 were admitted for some respiratory ailment and had no heart disease. The roentgenologic or clinical diagnoses in these cases were: bronchopneumonia—8; spastic bronchitis—7; lobar pneumonia—5; bronchitis—3; and bronchiolitis, viral pneumonitis, and aspiration pneumonia—1 each. Ten patients were cardiac cases, although most of them were studied roentgenographically for a suspected pulmonary infection. The majority of these (5 cases) had congenital heart disease with signs of left-to-right shunt. The remaining had: tetralogy of Fallot—1, pericardial effusion—2, left aberrant coronary artery—1, and 1 case was labelled as idiopathic cardiomegaly. In 1 instance, the vertical fissure line was an incidental finding in a newborn child with idiopathic megacolon; there was an enormously distended abdomen and in the chest there were signs of incomplete aeration of the lungs.

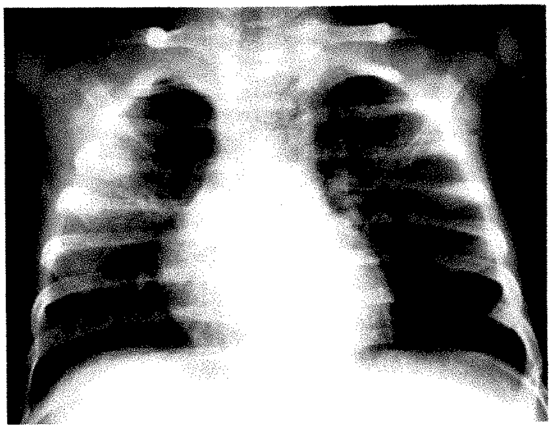

Fig. 1. Male, 6 months of age, under treatment with antibiotics for bilateral bronchopneumonia. Control roentgenogram shows vertical line on the right side.

Thirty-three of the total number were 1 year or less of age, the remaining were 2, 3, 5 and 6 years old, respectively.

In 26 cases the vertical line was seen on the right side, in 7 on the left and in 4 on both sides. Of the latter 4 cases, 3 had cardiac lesions and the fourth had Hirschsprung's disease.

Although the finding on the original roentgenograms is obvious, it cannot always be recognized on reproductions in print; however, Figures 1 through 3 show 3 characteristic cases.

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* Eleven cases briefly reported elsewhere (1964) are included.

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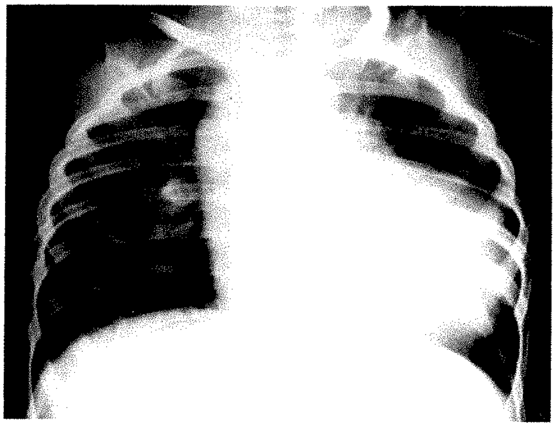

FIG. 2. Male, 2 years of age, with congenital heart disease. Vertical line is seen on the left side.

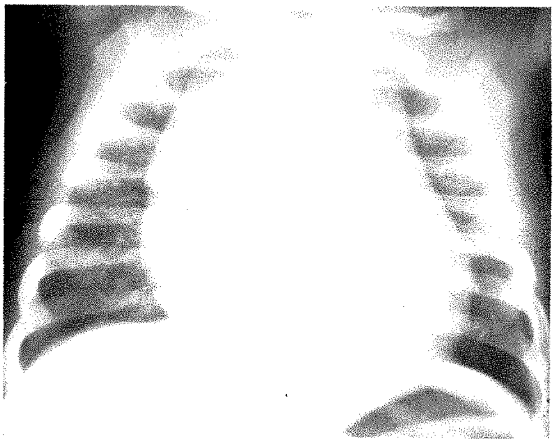

FIG. 3. Male, 11 months of age, with congenital heart disease. Bilateral vertical lines are demonstrated.

DISCUSSION

Davis considers the vertical line to be the lateral portion of the major fissure, rotated into the sagittal plane by the enlarged heart. Webber and O'Loughlin also consider it to be the fissure line, simply as a result of variation in its position. The first explanation, however, does not apply to patients with normal hearts, as Davis pointed out himself, and the second can hardly be accepted, as the finding is inconstant even in the same patient. It, therefore, appears that there should be some reason for such a temporary variation.

In the author's opinion, the vertical line represents the lateral border of a partially collapsed lower lobe, as it is projected by the tangential ray. Figure 4, *A* and *B* shows

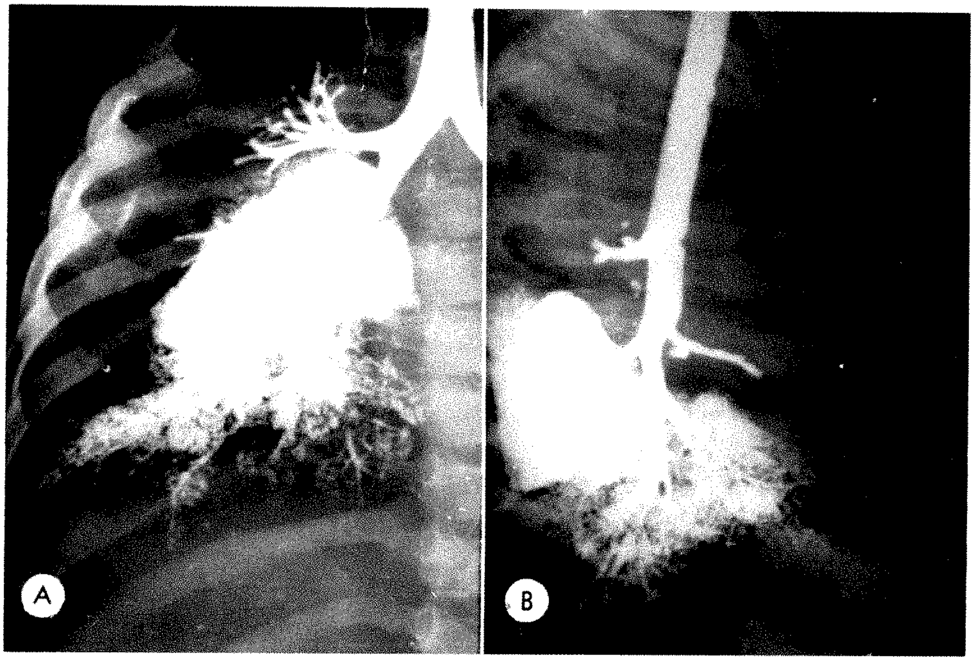

FIG. 4. Postmortem (*A*) anteroposterior and (*B*) lateral bronchograms in a case where a vertical line was seen before death. The right lower lobe is partially collapsed and retracted medially and posteriorly.

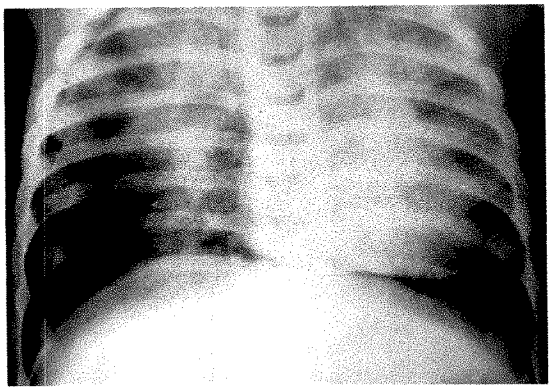

Fig. 5. Right pneumothorax. The border of the partially collapsed lower lobe corresponds in form and localization to the described vertical fissure line.

postmortem bronchograms of a patient in whom the vertical line was visualized during life, and is still seen faintly on the original postmortem roentgenogram. The bronchi of the lower lobe are crowded and the lower lobe is displaced medially and posteriorly. Figure 5 shows a partially collapsed lung in a case of pneumothorax, and the lateral margin of the lower lobe has the same form and direction as the discussed line. A similar line, only somewhat more medially situated, is considered by Caffey[1] as evidence of partial atelectasis in the lower lobe. Further evidence of difference in aeration between the lower lobe and the adjacent ones is the frequently observed hyperlucency of that part of the lung situated laterally to the line as compared to the area on the medial side.

Cardiomegaly alone could cause this difference in aeration, as the lower lobe bronchi are nearest to the enlarging heart. It may also be that bronchopulmonary infection is a common denominator for all the cases. It is well known that emphysema and pneumonitis are common findings in congenital heart disease.[3,5]

In 1 patient of whom 2 roentgenograms were made in an interval of a few minutes, we observed the line only on 1 roentgenogram, and this was in an inspiratory phase; the other was an expiratory study. The relative rarity of inspiratory chest roentgenograms in infants and small children might explain the infrequent observation of Davis' line, even in those cases in which it might be expected.

CONCLUSION

In the author's opinion, there is enough evidence to support the view that Davis' vertical line represents the lateral margin of the partially collapsed lower lobe. It is suggested that it has diagnostic value as a further sign of local respiratory impairment in the absence of other signs.

SUMMARY

Thirty-seven cases of a vertical line visualized in the roentgenograms of the lungs in infants and small children, first described by Davis, are discussed. Evidence is reported to support the view that the presence of this line points to impaired ventilation of the lower lobe of the lung.

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PNEUMOCYSTIS CARINII PNEUMONIA*

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THE first case of Pneumocystis Carinii pneumonia was reported in this country by Lunseth *et al.*[16] in 1955. Since then a number of cases have been reported in the American medical literature. Because the roentgen examination of the chest may play a role in the presumptive diagnosis of the disease, 6 cases are discussed (Table 1) with emphasis on the roentgen manifestations.

ETIOLOGY

Pneumocystis Carinii pneumonia is believed to be caused by the organism, Pneumocystis Carinii, which is thought to be a protozoon but may possibly be a fungus and occurs predominantly in patients in whom there is an alteration in the immune mechanism. The disease was observed first in Central Europe in epidemic form in institutionalized infants 2 to 4 months of age who were born prematurely or who were debilitated. Since then, cases have been reported from all parts of the world in both epidemic and endemic form. In the United States, Canada, and England, Pneumocystis Carinii infection appears endemic and has been associated with disturbances of the immune mechanism such as hypogammaglobulinemia,[6,16] Wiskott-Aldrich syndrome,[22] cytomegalic inclusion disease,[10] leukemia and lymphomas,[23] and more recently osteopetrosis.[1] It has been reported in children of all ages and in adults.[22] The increased incidence at 2 to 4 months of age, as reported in the European literature, coincides with that period in life when the gamma globulins are believed to be at their lowest physiologic level.

The protozoon is a saprophyte found in the lungs of many animals, *e.g.*, the rat, the mouse, the rabbit, the dog.[2] It was first reported by Chagas in 1909, and then by Carini in 1910, for whom the organism was named.[8] The characteristic pulmonary pathology was first described in 1938 by Ammich in Europe, although the clinical state had been recognized earlier in Germany. Vanek, in 1951, described Pneumocystis Carinii in the lungs of infants who had the clinical and histologic features of plasma cell pneumonia and considered this organism to be the probable etiologic agent,[16] although it has not been possible to confirm fully Koch's postulates. The proof that Pneumocystis is the causal agent in plasma cell pneumonia to date is indirect, but, nevertheless, rather convincing, as the organism has been found in all patients with this disease. The organism can be identified in microscopic sections of tissue by utilizing various staining techniques; *e.g.*, the McManus-Hotchkiss method; Masson's trichrome method; and with Giemsa as well as with Rio-Hortega's silver stain.[2] Sheldon[20] was able to stimulate the growth of the organisms already present in the lungs of rabbits by the administration of cortisone which he thought produced an alteration in the host-parasite relationship.

The organism has been noted as an incidental finding in the lungs of children and adults who have died of unrelated causes, which suggests that Pneumocystis can be present in human lungs in the absence of clinical symptoms until an alteration in the resistance of the host permits the disease to evolve and become clinically manifest.[17]

PATHOLOGY

The lungs are large, heavy, firm, pearl gray, and almost airless. Histologically, the lungs show an extensive mononuclear cellular infiltration. In the reports from Europe, the interstitial cellular infiltrate has been

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Table I

Patient	Age	Race	Sex	Underlying Disease	Treatment	Survival
1. R.S.	4½ mo.	N	F	Wiskott-Aldrich syndrome	Tetracycline Prednisone Vancomycin	Expired 4 days after admission
2. P.S.	6 mo.	W	M	Hypogammaglobulinemia	Gammaglobulin Tetracycline Methecillin	Expired 19 days after admission
3. J.S.	4 mo.	W	M	None demonstrated	Tetracycline Dexamethasone	Expired 4½ months after admission
4. E.V.	6 yr.	W	F	Acute lymphatic leukemia	Prednisone Gammaglobulin Penicillin	Expired 4 days after admission
5. M.McD.	6 mo.	W	F	Hypogammaglobulinemia	Gammaglobulin Hydroxystilbamine	Expired 3 days after admission
6. A.G.	10 mo.	W	M	Hypogammaglobulinemia	Gammaglobulin Hydroxystilbamine Penicillin Tetracycline	Expired 1½ months after admission

Note: All patients were proven to have Pneumocystis Carinii pneumonia by biopsy of the lung and/or at postmortem examination (see Figures 1 through 6).

described as being composed predominantly of plasma cells, but this has not been the case in this country or in those countries outside of Europe. Some believe these to be histiocytes and/or cells intermediate between plasma cells and histiocytes. There are few polymorphonuclear leukocytes and lymphocytes. The alveoli are filled with a characteristic foamy or "soap bubble" exudate in which the organisms can be identified.[10]

Electron microscopy of ultra-thin sections of alveolar fluid from human lungs infected with Pneumocystis Carinii has demonstrated the presence of mature cysts with characteristic nuclear-like "cyst bodies" as well as many crescent forms which seem to be typical for human Pneumocystis infections. The cysts have a thick wall with an inner and outer membrane. Each cyst

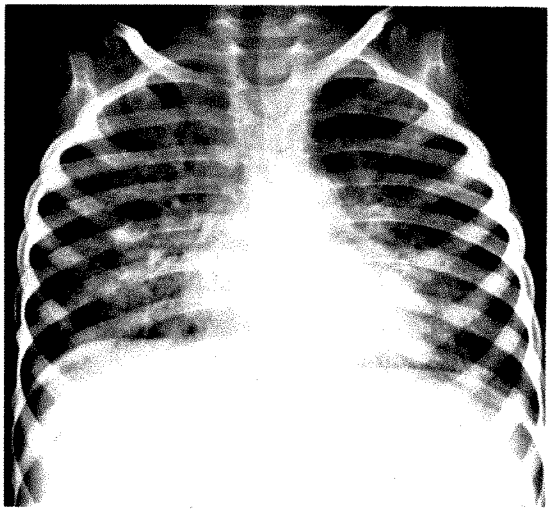

Fig. 1. R.S., female, 4½ months of age. There are diffuse interstitial and peribronchial infiltrates throughout both lungs with peripheral and interstitial emphysema. Neither hilar lymphadenopathy nor pleural disease is present.

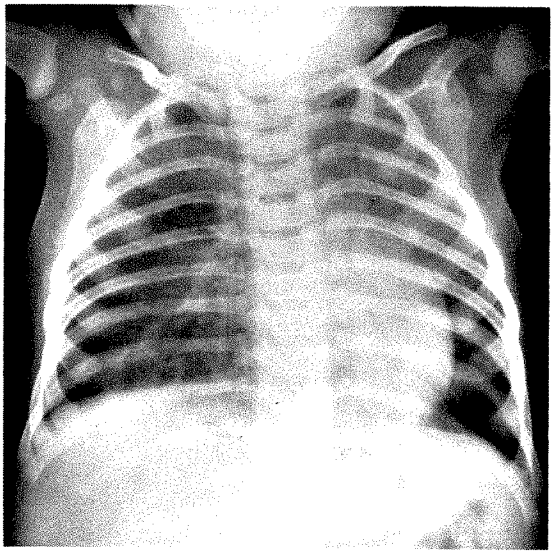

FIG. 2. P.S., male, 6 months of age. The lungs are over-aerated and there is extensive parenchymal disease. An air bronchogram is seen behind the heart on the left. The diffuse density of the lungs is consistent with an intra-alveolar exudate. Volume loss involving the left lung is manifested by the shift of the heart and mediastinum to the left.

contains several nuclear-like bodies. These bodies have a complex lamellar or tubular structure in their interior and seem to be enveloped in a distinct membrane which has two or more contours.[5]

CLINICAL PICTURE

The most characteristic finding is tachypnea with peri-oral and peri-orbital cyanosis. In spite of the severe respiratory distress, the lungs are frequently normal to auscultation and percussion. Occasionally, fine crepitant rales on coughing or deep breathing, slightly impaired resonance, and bronchial breath sounds are evident over some areas of the lungs. This discrepancy between the severity of the pulmonary symptoms and the physical findings is typical. Fever is not usually a factor, the temperature rarely going above 100° F. unless there is a superimposed bacterial infection. The onset of the disease is usually slow and insidious with restlessness or languor and poor feeding. The duration of the disease is from 4 to 6 weeks, although at times only a few days elapse prior to death. The reported mortality rate varies from 20 to 50 per cent, but it is difficult to ascertain accurately as it is likely that there are sub-clinical cases.[2,10,11,19]

LABORATORY FINDINGS

Laboratory data are of limited help in making the diagnosis. The white blood cell

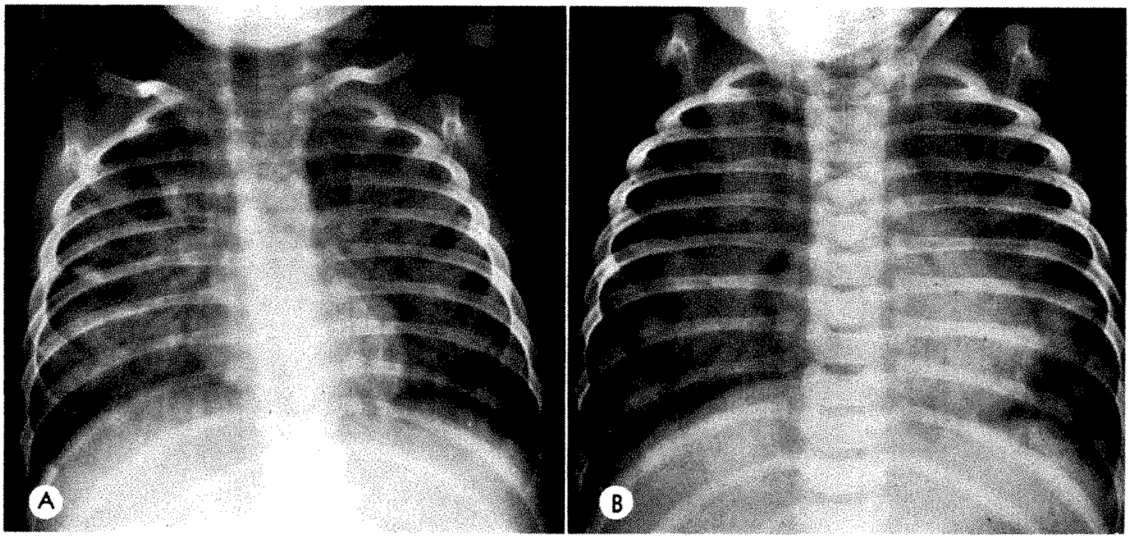

FIG. 3. J.S., male, 4 months and 7 months of age. (*A*) Peripheral and interstitial emphysema are present bilaterally as well as linear and patchy areas of abnormal density. There is no evidence of hilar lymphadenopathy or pleural disease. (*B*) Three months later, there is still peripheral and interstitial emphysema, but the parenchymal disease has become more confluent. All lobes are involved.

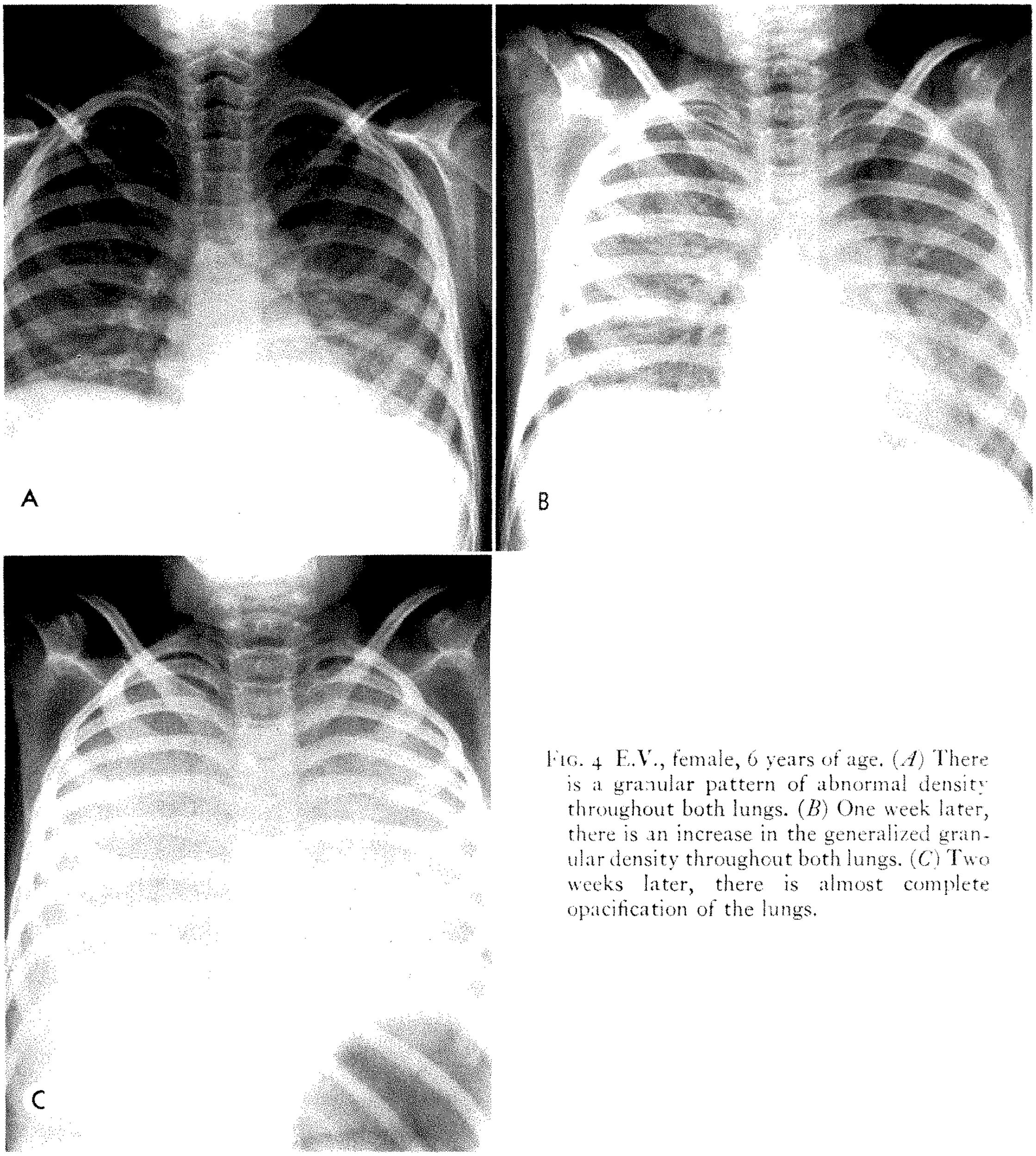

Fig. 4 E.V., female, 6 years of age. (*A*) There is a granular pattern of abnormal density throughout both lungs. (*B*) One week later, there is an increase in the generalized granular density throughout both lungs. (*C*) Two weeks later, there is almost complete opacification of the lungs.

count is normal or only slightly elevated and associated with a normal differential count or slight neutrophilia. Slight to marked eosinophilia has been observed. There have been reports of the plasma calcium levels being as high as 20 mg. per 100 ml., but this has not been a consistent finding. Plasma proteins are usually within the range of normal.[10] Abnormalities consistent with the underlying disease are to be found when Pneumocystis Carinii infection is secondary.

ROENTGENOGRAPHIC FINDINGS

The roentgenographic appearance of the chest is similar in all patients. There is a fine, almost homogeneous, granular pattern of density throughout the pulmonary parenchyma, which presents first in the hilar regions and then progresses to the periph-

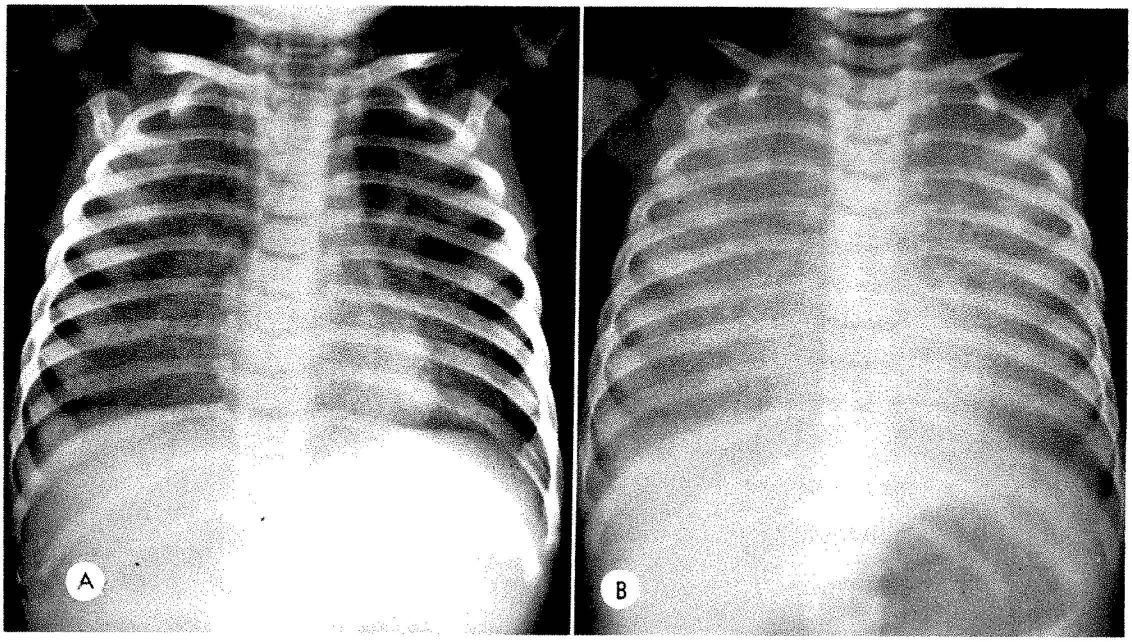

Fig. 5. M.McD., female, 6 months of age. (*A*) A granular, almost homogeneous density is present and involves both lungs. Air bronchograms are seen in both lungs. The lungs are over-aerated. Hilar lymphadenopathy and pleural disease are absent. (*B*) Nine days later, the lungs are almost homogeneous in their opacity.

ery (Fig. 4, *A*, *B* and *C;* and 5, *A* and *B*). The peripheral portions of the lungs are emphysematous (Fig. 3, *A* and *B*). In addition to interstitial emphysema, pneumothorax and pneumomediastinum may be present. The parenchymal involvement progresses toward the periphery to involve all of the lungs and to become more confluent until frequently the thorax is completely opacified. Usually, all lobes are involved, but not necessarily so; at times the infiltrates may be subsegmental in distribution with over-aeration of the remaining portions of lung (Fig. 1, 2, and 6). In spite of the extensive involvement of the lung, pleural reaction and hilar lymphadenopathy are absent. An air bronchogram is usually present as one would expect to find in the presence of intra-alveolar infiltrates of any etiology.[7,8,9]

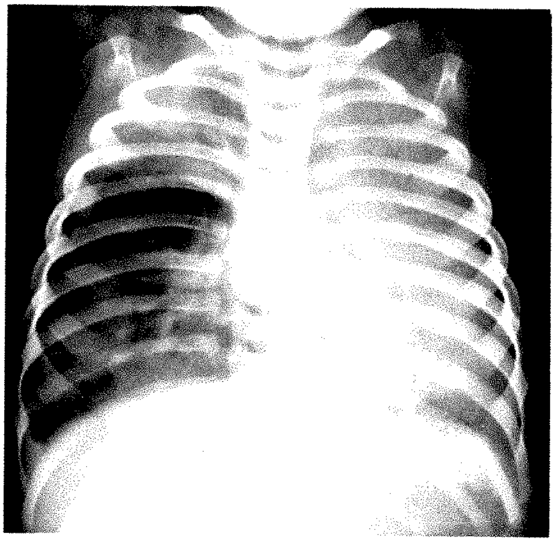

Fig. 6. A.G., male, 10 months of age. There is diffuse disease throughout all lobes with the exception of the right middle lobe which is emphysematous.

Atelectasis is not an uncommon feature and, in spite of obvious loss of volume of portions of the lung, emphysema will be evident as manifested by the low position of the diaphragm and the wide intercostal spaces (Fig. 2 and 6).

Roentgenographic abnormalities may be present early in the disease before signs of severe respiratory distress become manifest. Several patients proven to have Pneumocystis Carinii pneumonia have been cured of the disease and have been followed roentgenographically. The changes within the lungs were shown to clear within 6 weeks, although 1 patient has been reported in whom complete resolution did not take place until 2½ years after the onset.[8]

Other entities to be considered in differential diagnosis on the basis of the roentgen appearance are the reticuloendothelioses, idiopathic pulmonary hemosiderosis, giant cell pneumonia, alveolar proteinosis, and, in the early stages, lipoid pneumonia, lesions responsible for obstruction to pulmonary venous return, and, at times, the Hamman-Rich syndrome.

DIAGNOSIS

Though roentgenographic alterations are not specific with respect to the etiology, they should, when the clinical findings of progressive respiratory distress and cyanosis are taken into consideration, strongly suggest the diagnosis. The definitive diagnosis, however, is made by the demonstration of the pneumocysts. They may be recovered either from bronchial secretions or from biopsy of the lung. Since it may be difficult to demonstrate the pneumocysts in bronchial washings, biopsy of the lung offers the best method for diagnosis. This may be performed by either needle aspiration or open biopsy. Open biopsy is probably the preferred method because it results in a large sample of tissue for evaluation by the pathologist.[10]

A complement fixation test, utilizing extract from the lungs of affected infants, has been reported by Vivell, Buhn, and Lips in 1956. According to them, a titer of 1 to 4 may be regarded as positive for pneumocyst antibodies, but a rising titer is more conclusive. A positive reaction usually occurs during the second week of illness.[16]

TREATMENT

There is no specific treatment to date, though early reports on the use of the aromatic diamidines and pentavalent antimony compounds have shown some promise. There are not sufficient cases in which these drugs have been used to adequately estimate their value.[16]

Treatment has, for the most part, been directed toward reducing the symptoms with oxygen and mist, resulting in considerable relief from respiratory distress. Broad spectrum antibiotics are utilized when superimposed bacterial pneumonia is present. The use of cortisone has been deleterious and is believed to enhance the spread of the infectious process.

SUMMARY

Pneumocystis Carinii pneumonia most often occurs in debilitated infants and children or those with more specific alterations of the immune mechanism. The characteristic clinical picture is one of progressive respiratory distress and cyanosis, with relatively few physical findings pertaining to the thorax. The roentgen findings are those of diffuse, granular pulmonary infiltrates which progress from the hili toward the periphery of the lung, becoming more and more confluent. Interstitial emphysema may be present and, not infrequently, pneumothorax and pneumomediastinum. Pleural reaction and hilar lymphadenopathy are absent. Although the roentgen findings are not specific with respect to the etiologic agent, when they are correlated with the clinical picture, the presumptive diagnosis of pneumonia due to Pneumocystis Carinii may be made.

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THE ROENTGENOGRAPHIC FINDINGS IN INFANTILE AND CHILDHOOD HISTOPLASMOSIS*

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THE manifestations of histoplasmosis in the adult were described by Darling[3] in 1906, but it was not until 1934, twenty-eight years later, that a case of active infantile histoplasmosis was reported;[4] and today, though the abnormalities produced by this disease in the chest roentgenograms of adults have been thoroughly analyzed, there still has been little written about chest findings in active infantile and childhood histoplasmosis. Attention has centered upon pulmonary calcifications in relation to skin sensitivity to the histoplasmin antigen.[2]

The subject is complicated because different changes appear in localized and generalized histoplasmosis and because the changes in infants and children differ markedly from those seen in the adult.

ROENTGENOGRAPHIC EXAMINATION

In this report the significant findings in the chest roentgenograms of 47 patients with proven, active histoplasmosis are summarized. The patients were under 18 years of age and residents of Memphis and Shelby County where the percentage of positive histoplasmin skin reactors is remarkably high. Recent statistics[5] show that 88 per cent of the Caucasian and 92 per cent of the Negro navy recruits from this area have positive skin tests. The incidence of the childhood disease must, therefore, be extremely high. Nevertheless, the number of proven active cases recorded in the major Memphis hospitals is surprisingly small. In the 1960 to 1964 records of 5 hospitals and the 1953 to 1964 records of a sixth, a total of only 47 cases was found in which chest roentgenograms were made.

The striking difference between the number of positive skin reactors and the number of proven active cases indicates that childhood histoplasmosis, in the vast majority of cases, resolves subclinically.

In the 47 cases selected, diagnosis was established either (1) by identification of *Histoplasma capsulatum* in the blood, bone marrow, surgical specimen, or at autopsy, or (2) by significant serologic titer, a positive histoplasmin and a negative tuberculin skin test, and the clinical exclusion of other diseases. Nineteen of the patients were in the first group, 28 in the second. The large number of cases that had only a positive skin test and a presumptive clinical diagnosis was excluded.

The clinical records and roentgenograms were reviewed and the results tabulated under multiple categories including both clinical and roentgenographic findings.

RESULTS

Of the 47 patients, 16 were under 1 year of age. All of these had generalized disease and 12 died soon after admission. Out of 10 infants in this group who were skin tested only 1 had a positive reaction. The chest roentgenograms showed essentially clear lung fields and only 2 of the 16 manifested hilar lymphadenopathy. Apparently these infants died before gross lesions could develop, because autopsies on some disclosed the organism throughout the lung parenchyma in large numbers. In 11 of these 16 cases, splenic enlargement was evident in the chest roentgenograms.

Only one patient fell in the age group of 1 to 4 years. The other 30 patients were distributed evenly through the age group of 4 to 17 years. In these cases as the age increased, the disease tended to become more localized. The most consistent finding

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TABLE I

ROENTGENOGRAPHIC AND CLINICAL MANIFESTATIONS ACCORDING TO AGE

	No. of Cases	Localized	Mildly Generalized	Generalized	Died	Positive Skin Test	Positive Serology	Clinical Splenomegaly	Organism Demonstrated	Negative Lungs	Pneumonia	Calcification	Hilar Lymphadenopathy	Roentgenographic Splenomegaly
4 mo.–1 yr.	16	0	2	14	12	1 (of 10)	2 (of 4)	16	15	10	6	0	2	11
2 yr.–3 yr.	1	0	1	0	0	1	1	0	0	0	0	0	1	1
4 yr.–6 yr.	10	6	4	0	0	9	10	3	0	0	5	4	10	5
7 yr.–18 yr.	20	18	1	1	0	20	17 (of 18)	2	4	1	11	10	17	3
Total	47	24	8	15	12	31	30 (of 33)	21	19	11	22	14	30	20

TABLE II

MANIFESTATIONS ACCORDING TO CLINICAL TYPES

	No. of Cases	Age Range	Severely Ill	Roentgenographic Lung Changes	Clinical Organomegaly	Hilar Lymphadenopathy	Death
Generalized	15	4 mo.– 7 yr.	14	6	15	3	12
Mildly Generalized	8	4 mo.–12 yr.	0	6	7	6	0
Localized	24	4 yr. –17 yr.	0	24	2	19	0

was hilar lymphadenopathy which was found in 28 patients. A prominent right paratracheal lymph node was present in 10 of these. Esophageal and bronchial compression from hilar lymphadenopathy was present in 8 and is, therefore, probably more common than generally recognized. One case showed fibrosing mediastinitis around the hilar lymphadenopathy. Seven cases manifested a widespread pulmonary infiltration with varying amounts of confluence. This usually occurred in the older children.

No case showed significant pleural involvement.

There was no significant seasonal variation; and in contrast to the predilection of

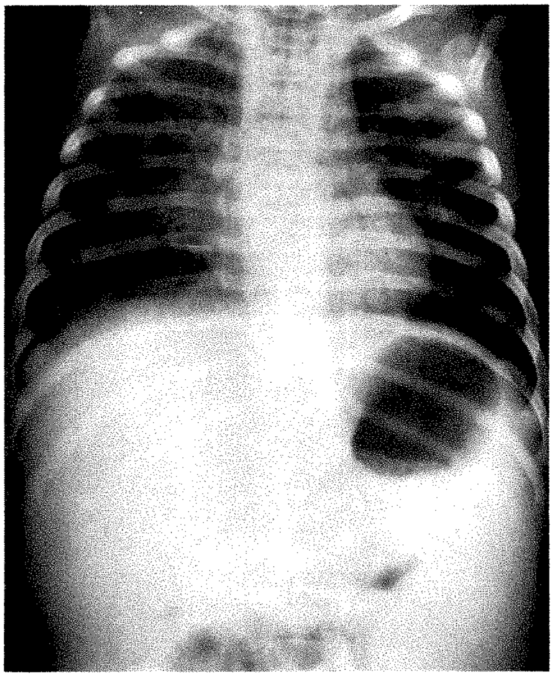

FIG. 1. Four month old white male admitted with fever for 1 week and hepatosplenomegaly. Histoplasmin skin test was negative but bone marrow culture showed *Histoplasma capsulatum.* Chest roentgenogram reveals only a questionable infiltrate in the right base but definite evidence of hepatosplenomegaly.

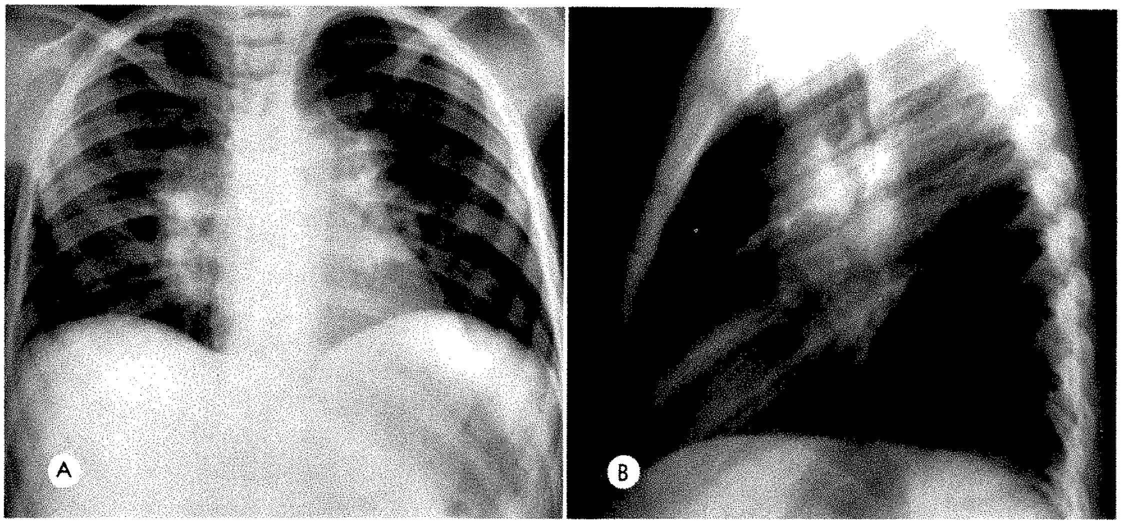

Fig. 2. (*A* and *B*) Bilateral hilar lymphadenopathy in a 5 year old Negro female with fever, cough and anorexia for 1 week. Histoplasmin skin test was positive and complement fixation was positive with a titer of 1:256.

adult histoplasmosis for the elderly male, the cases were divided evenly between the sexes.

There were no deaths in the children over 1 year of age; and, in general, symptoms subsided in a matter of weeks. Surgery was performed in 8 cases, either for diagnostic purposes or for relief of lymph node compression.

Table I shows the roentgenographic and clinical manifestations according to age. In Table II the cases are classified according to type of clinical disease. Figures 1 through 5 demonstrate some of the roentgenographic findings.

DISCUSSION

It is not to be concluded that these cases are representative of all cases of active childhood histoplasmosis. Our series includes only the small percentage with symptoms severe enough to require hospitalization. Only cases that had roentgenograms were included and, therefore, this eliminated several cases of fulminant infantile disease which died before roentgenograms could be obtained.

It appears that the generalized infantile form is a completely different disease than the more localized subacute form seen in the older child. One of the prominent findings on chest roentgenograms in the infant group was an enlarged spleen. Splenomegaly in an acutely ill infant should suggest histoplasmosis. The majority of infantile cases was free of lung parenchymal changes on admission and, in the few that did have basilar pneumonia, this may well have

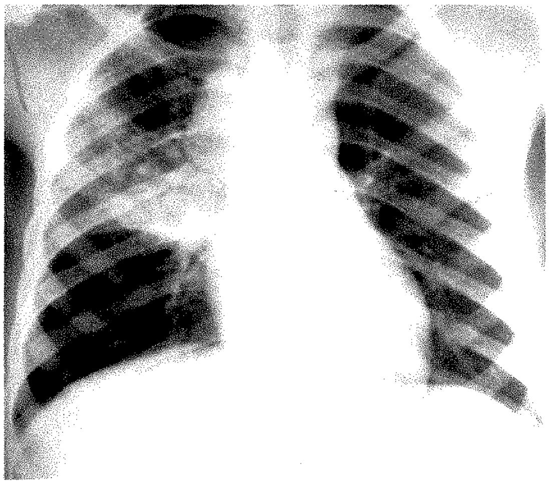

Fig. 3. Large right paratracheal lymph node and pneumonia in a 9 year old Negro male with cough and chest pain for 3 days. Complement fixation showed 1:256 positive titer and hemagglutination test was positive with titer of 1:128.

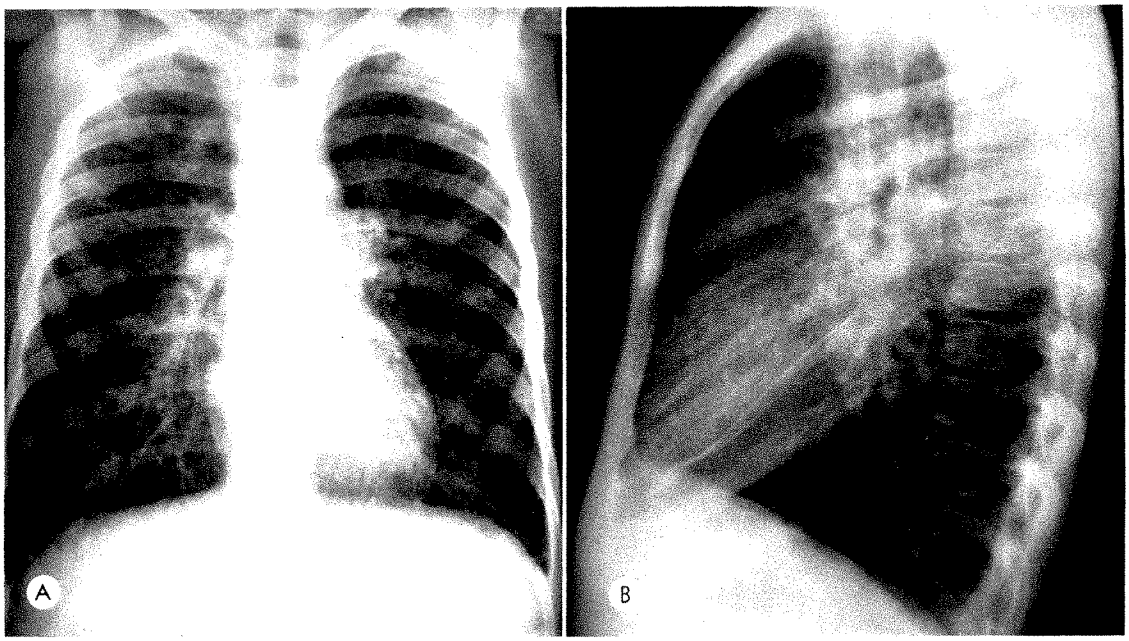

FIG. 4. (*A* and *B*) Widespread pneumonia and hilar lymphadenopathy in a 10 year old white male who began having fever and cough 5 days after playing in a cave. Skin test, complement fixation and hemagglutination tests were all strongly positive.

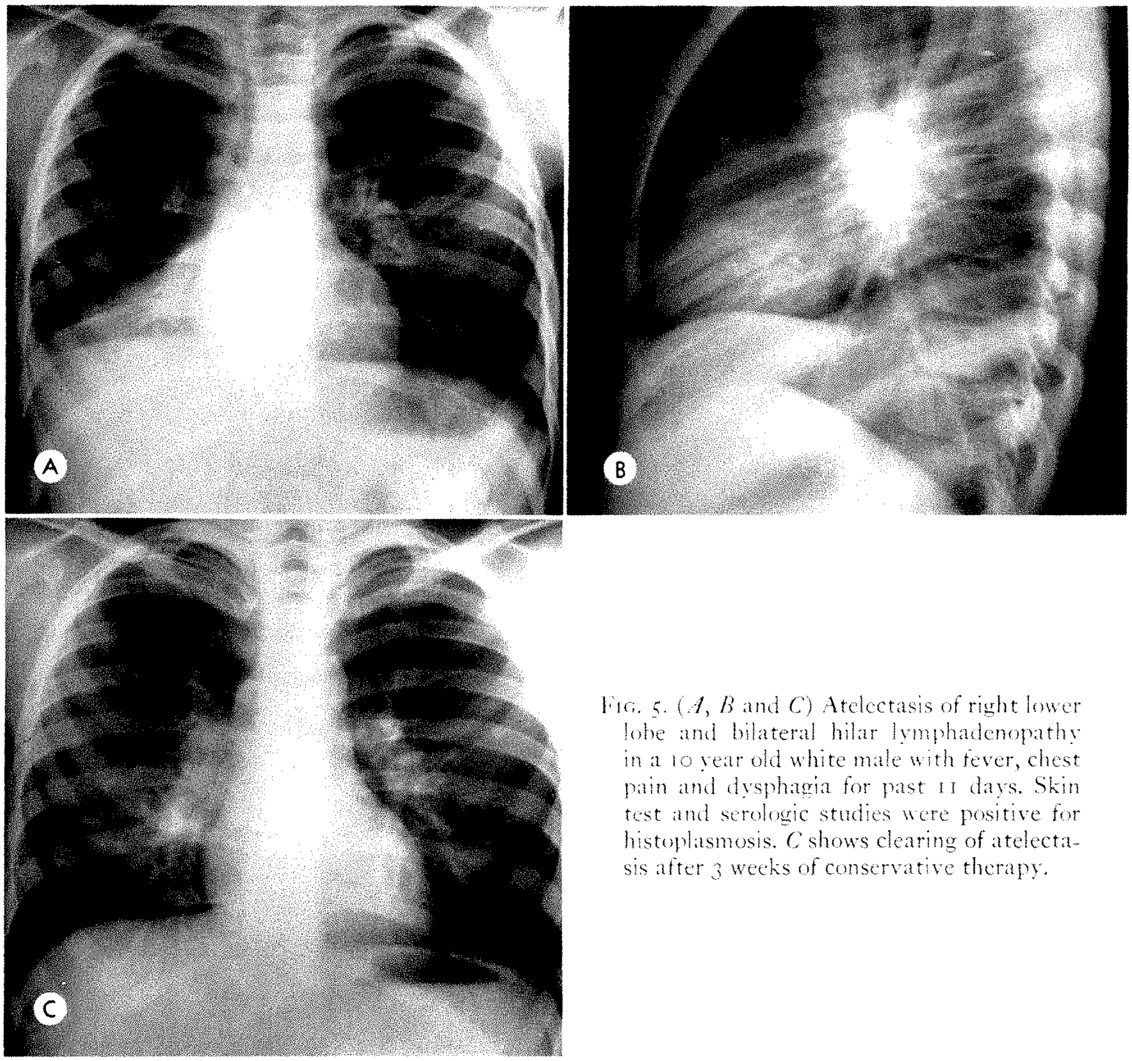

FIG. 5. (*A*, *B* and *C*) Atelectasis of right lower lobe and bilateral hilar lymphadenopathy in a 10 year old white male with fever, chest pain and dysphagia for past 11 days. Skin test and serologic studies were positive for histoplasmosis. *C* shows clearing of atelectasis after 3 weeks of conservative therapy.

been due to secondary nonspecific infection in a severely ill child. It should be remembered that a negative skin test and chest roentgenogram do not exclude acute histoplasmosis in an infant.

It is interesting to note that there was only 1 case in the age group between 1 and 4 years. Perhaps there is a decreased incidence of active manifestation in this age group.

No case manifested the chronic cavitary upper lobe process typical of the elderly Caucasian male. In the older children, the sequence of diffuse pulmonary infiltration, patchy pneumonia and ultimate calcific scarring paralleled the course of the infection in adults.

SUMMARY

Forty-seven cases of active, proven, childhood histoplasmosis have been reviewed and tabulated according to age and clinical type of infection.

Histoplasmosis presents a variety of findings depending upon whether the infection is acute, subacute, chronic, localized or generalized. The course of the disease in infants and children differs greatly from that in the adult.

In the cases reviewed, infants under 1 year of age tended to have an acute, generalized, fatal infection with hepatosplenomegaly and negative skin tests. Of the 16 cases, 11 had essentially clear lung fields on admission and only 2 showed hilar lymphadenopathy. An enlarged spleen was evident on the chest roentgenograms in 11.

Twenty-eight of the 31 other children had hilar lymphadenopathy, and in 8 cases enlarged lymph nodes compressed a bronchus and/or the esophagus. In 7 of these 31 children, there was a widespread pulmonary infiltration with some degree of patchy confluence.

In contrast to the findings in adults, neither cavitation nor pleural involvement was seen in either the infants or the children.

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We wish to express our appreciation to Dr. George Cooper, Jr., the Chairman of our Department, for his most helpful advice in preparing this report.

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TRAUMATIC LUNG CYST*

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THE finding of 3 discrete, acutely formed, lung cysts on the roentgenograms of 3 different patients who had sustained nonpenetrating chest trauma prompted a search of the available literature for information regarding this entity.

That significant and various types of thoracic injury—that is injury to the chest wall, the pleurae, the lung parenchyma, and the mediastinal contents—result from nonpenetrating chest trauma is amply documented in the medical literature.[1,2,3,15–21] Such entities as fractured ribs, pneumothoraces, hemothoraces, and a combination of any 2 or all 3 represent well-known and readily recognizable manifestations of nonpenetrating chest trauma.

Other medically publicized entities which are peculiar to nonpenetrating chest trauma but are perhaps less often appreciated and recognized are pulmonary hematoma, pulmonary laceration, and pulmonary contusion.[9,17–21] Atelectasis and emphysema of varying degrees and in various locations also result from closed chest trauma.[9,21]

Acknowledgement that lung cysts can be associated with nonpenetrating chest trauma is also made by several authors.[2,3,5,8,17–21] In dealing with this latter entity—traumatic lung cysts—there has been a tendency to regard these cysts as secondary lesions; *i.e.*, cavities or pneumatoceles resulting from the resolution of a pulmonary hematoma. To be sure, the resolution of a pulmonary hematoma may result in a residual pulmonary cyst or cavity. However, as will be illustrated and emphasized in this paper, many traumatic lung cysts are acutely formed primary, not secondary, structural manifestations of nonpenetrating chest trauma. They represent still another entity peculiar to closed chest trauma.

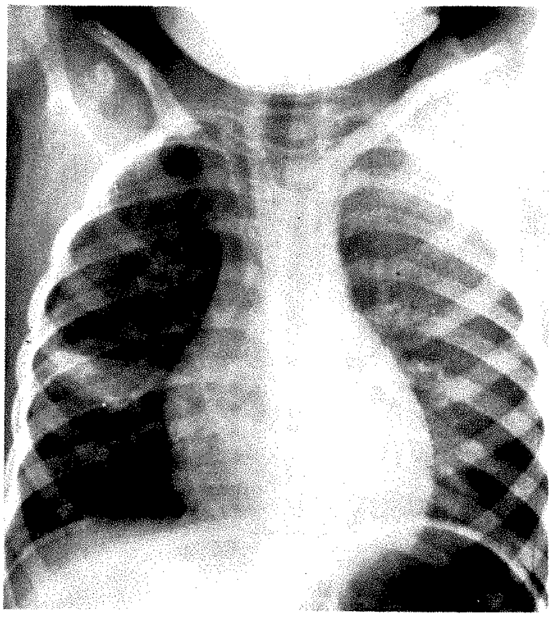

FIG. 1. Case 1. Posteroanterior roentgenogram of the chest, taken 1 hour after the patient had been struck by a bus, shows a small pneumothorax and a 2.5 cm. long, mid lung field, air fluid level on the right. Nonpenetrating chest trauma had occurred on the left.

REPORT OF CASES

CASE 1. E.V., a 4 year old female, was admitted to Coco Solo Hospital on February 8, 1964, twenty minutes after being struck by a bus. Multiple contusions and abrasions over the left posterior chest and flank surfaces represented the significant physical findings. During the physical examination, the patient coughed up some clotted blood. Vital signs were normal. Hemoglobin and hematocrit determinations were normal.

A roentgenogram of the chest dated February 8, 1964 (Fig. 1) and taken approximately 1 hour after the patient had been injured showed the left lung field to be clear and fully expanded; the left thoracic cavity was free of any observable fluid. The mediastinal structures presented a normal appearance and were located in the

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The contents of this paper reflect the personal view of the author and are not to be construed as a statement of official U. S. Army policy.

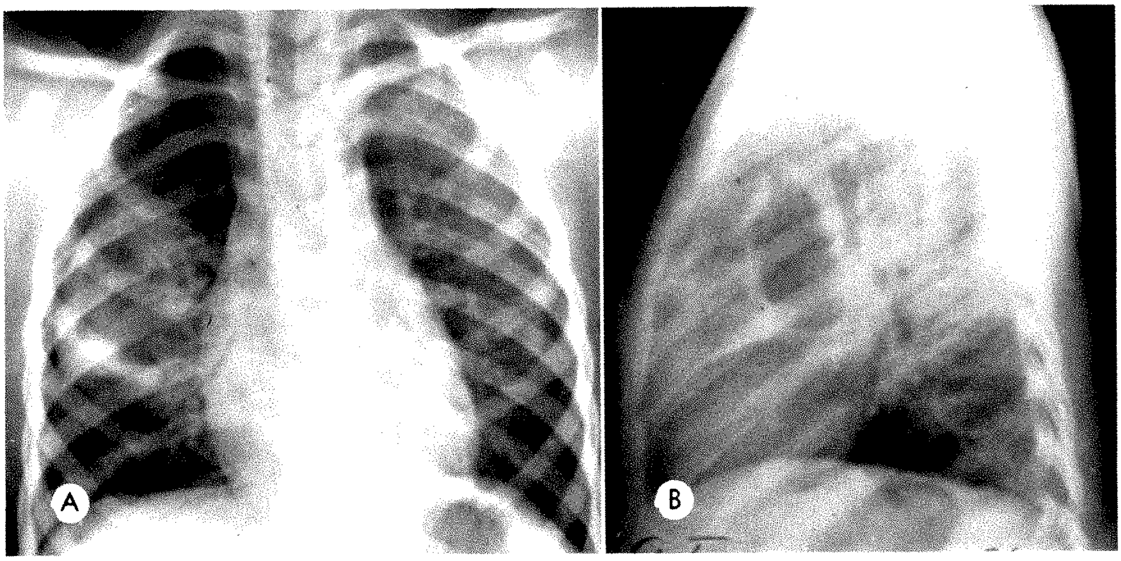

FIG. 2. Case I. (*A*) Posteroanterior and (*B*) right lateral roentgenograms of the chest, taken 24 hours later, demonstrate an increase in the size of the right pneumothorax. A large 2.5×2×4 cm., oval, multiloculated, traumatic lung cyst with a surrounding pulmonary reaction (contused lung) is present. The air fluid level seen on the roentgenogram taken 1 day earlier is absent.

midline. On the right side, a small pneumothorax was present; an associated rib fracture was not identified. A 2.5×3.5 cm., oval, cystic lesion containing an air fluid level was noted in the anterior segment of the right upper lobe. The rest of the incompletely expanded lung was clear. A normal chest roentgenogram taken 4 months earlier was available for comparison.

In view of the roentgenographic findings, the patient's parents were again questioned about any pre-injury symptoms or complaints referable to the respiratory tract. They were emphatic about their daughter's pre-accident good health.

Repeat roentgenograms taken 24 hours later on February 9, 1964 (Fig. 2, *A* and *B*) showed an increase in the size of the pneumothorax but no shift of the mediastinal structures. The lung cyst had enlarged and measured 2.5×2×4 cm. Its periphery was now surrounded by a 3 mm. thick parenchymal reaction. The cyst was multiloculated and no longer contained an air fluid level.

Roentgenograms taken 5 days post injury (not shown) demonstrated almost complete reexpansion of the right lung. The traumatic lung cyst was smaller; the previously associated pulmonary reaction had resolved.

A fourth set of roentgenograms were taken on February 24, 1964 (Fig. 3). Both the pneumothorax and the traumatic lung cyst were now absent. The minor fissure was thickened. The patient was discharged 2 days later.

Comment. The patient suffered closed chest trauma to the left side. Yet, a traumatic lung cyst, which was present on a roentgenogram taken only 1 hour after the

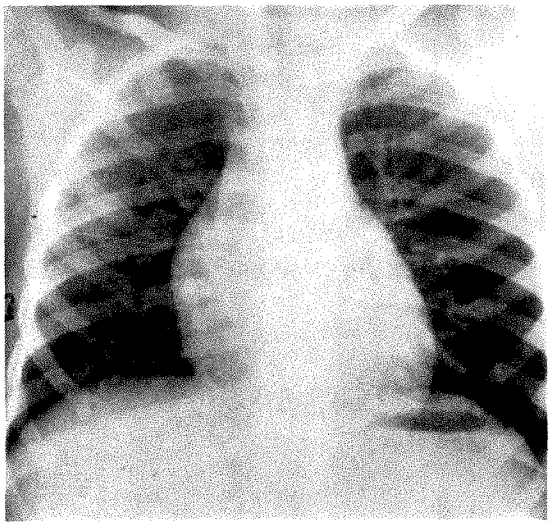

FIG. 3. Case I. Follow-up posteroanterior chest roentgenogram, taken 16 days post injury, demonstrates complete resolution of the traumatic lung cyst. There is residual minor fissure thickening.

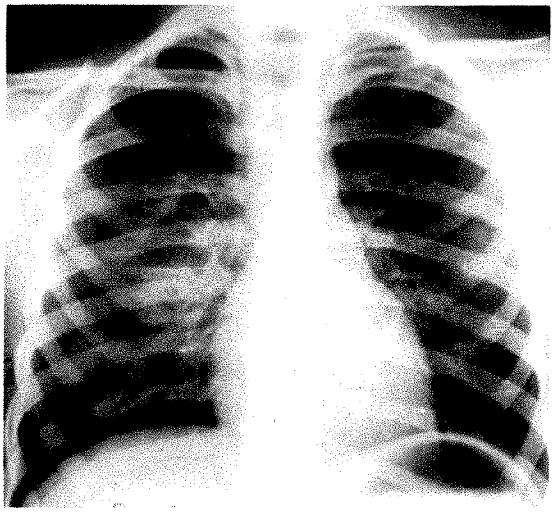

Fig. 4. Case II. Anteroposterior roentgenogram of the chest, taken with the mobile unit 4 hours after the patient had been struck by a car sustaining bilateral nonpenetrating chest trauma, demonstrates a small pneumothorax and 2×3 cm., oval, air fluid containing traumatic lung cyst on the right. A lateral chest roentgenogram (not shown) made with the mobile unit localized the traumatic cyst to the proximal portion of the anterior segment of the right upper lobe.

injury, developed on the right side—an example of a contrecoup injury and acute development of a traumatic lung cyst. Another interesting facet in this case was the possible association of clinical hemoptysis with the early evacuation of the air fluid level (in less than 24 hours) from the traumatic lung cyst.

Case II. G.B., a 6 year old female, was admitted to Gorgas Hospital on August 31, 1964 after being struck by an automobile 2 hours earlier. The patient was previously well. Fairly extensive but superficial abrasions over the posterior chest, and contusions over the right anterior chest and a small area of the left anterior chest were apparent on physical examination. The lungs revealed slight hyperresonance and diminished breath sounds over the right upper lobe posteriorly and coarse rales were audible in the right anterior chest. Vital signs were normal. A complete blood cell count was unremarkable.

An admission chest roentgenogram on August 31, 1964 (Fig. 4), taken 4 hours post injury with a mobile unit demonstrated the left lung field to be fully expanded and clear. The cardiac shadow was located in the midline and presented a normal appearance. On the right side, a small pneumothorax was present; an associated rib fracture was not demonstrated. A 2×3 cm. cyst containing an air fluid level and surrounding pulmonary reaction were demonstrated in the mid lung. A previously normal chest roentgenogram dated December 4, 1961 was available for comparison.

A right lateral chest roentgenogram (not shown) obtained with the mobile unit the next day localized the traumatic lung cyst in the anterior segment of the right upper lobe. The air fluid level was smaller but still present.

A chest roentgenogram taken 6 days later on September 7, 1964 (Fig. 5) demonstrated resolution of the previously described right pneumothorax. The cystic lesion was smaller, measuring 1.5×3 cm.; the surrounding reaction and air fluid level were now absent.

The patient was discharged on September 9, 1964. During the 10 day hospitalization, there was no clinical evidence of any respiratory difficulties. The skin abrasions healed, the right pneumothorax resolved, and the traumatic lung cyst had partially disappeared.

A follow-up chest roentgenogram taken 16 days post injury on September 16, 1964 (Fig. 6) demonstrated resolution of the lung cyst. An irregular shaped 3×4 cm. radiodensity believed to represent the collapsed wall of the former cyst or possibly a residual pulmonary hematoma was present and located at the same site as the former lung cyst.

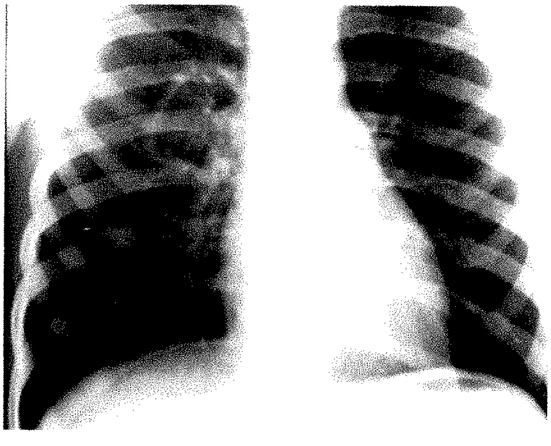

Fig. 5. Case II. Posteroanterior roentgenogram of the chest, taken 6 days later, shows reduction in the size of the traumatic lung cyst to 1.5×3 cm. The air fluid level previously associated with the cyst and the pneumothorax have resolved.

An additional follow-up chest roentgenogram on October 17, 1964 (Fig. 7) demonstrated no abnormality. The traumatic lung changes had resolved.

Comment. The admission chest roentgenogram—taken just 4 hours after the closed chest trauma—demonstrated a traumatic lung cyst. This time sequence, and the even shorter interval between the closed chest injury and the formation of a traumatic lung cyst in Case I, supplies objective evidence that traumatic lung cysts are in some instances acutely formed primary—not secondary—structural manifestations of nonpenetrating chest trauma.

Case III. L.G., a 17 year old male, was admitted to Gorgas Hospital on December 23, 1964, two hours after receiving an electric shock from a high voltage source and falling from a 40 foot electric pole. The patient, while being examined in the emergency room, vomited a large volume of semi-clear fluid which contained particles of clotted blood. There were no chest or respiratory findings. A 10 cm. laceration of the right thigh and electrical burns of both thighs and both hands represented the pertinent physical findings. The vital signs were normal and stable.

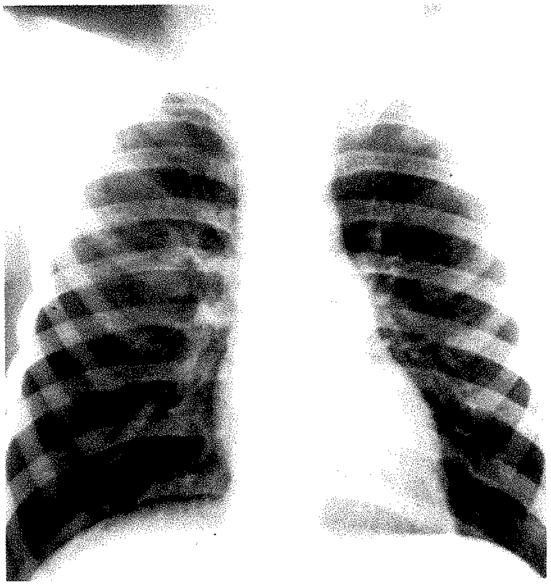

Fig. 6. Case II. Posteroanterior roentgenogram of the chest, taken 16 days post injury, demonstrates absence of the traumatic lung cyst. A discrete 1.2×2.5 cm. radiodensity, believed to represent the collapsed wall of the former cyst or a residual hematoma, is located at the site of the former cyst.

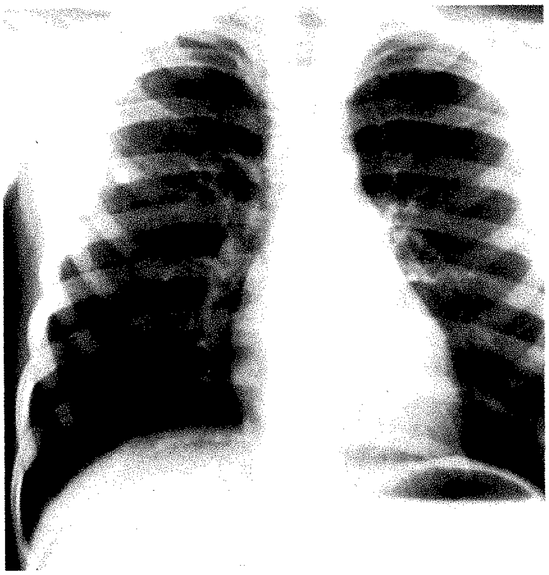

Fig. 7. Case II. Follow-up posteroanterior chest roentgenogram, taken 1 month later, demonstrates no abnormality. The traumatic lung changes have resolved.

Admission chest roentgenograms on December 24, 1964 (Fig. 8, *A* and *B*), taken 10 hours post injury, demonstrated no abnormality on the right side. The mediastinal structures appeared normal; there was no mediastinal air. On the left, a faint but definite air fluid level was projected over the heart shadow. A left lateral chest roentgenogram (Fig. 8*B*) clearly demonstrated the air fluid level and localized it in the posterior basal segment of the left lower lobe. The lung markings adjacent to the air fluid level were increased as if they were being compressed by some adjacent parenchymal expanding lesion. A left pneumothorax was not present.

Prior to procurement of the chest roentgenograms, the patient vomited fluid containing blood clots as he had done in the emergency room upon admission. One observer, who attempted to pass a nasogastric tube in this very emotionally upset patient, recorded the impression that the vomited clotted blood was swallowed blood, but a repeat physical examination of the upper respiratory tract failed to identify a bleeding point.

Well penetrated roentgenograms of the chest in the posteroanterior and left lateral upright

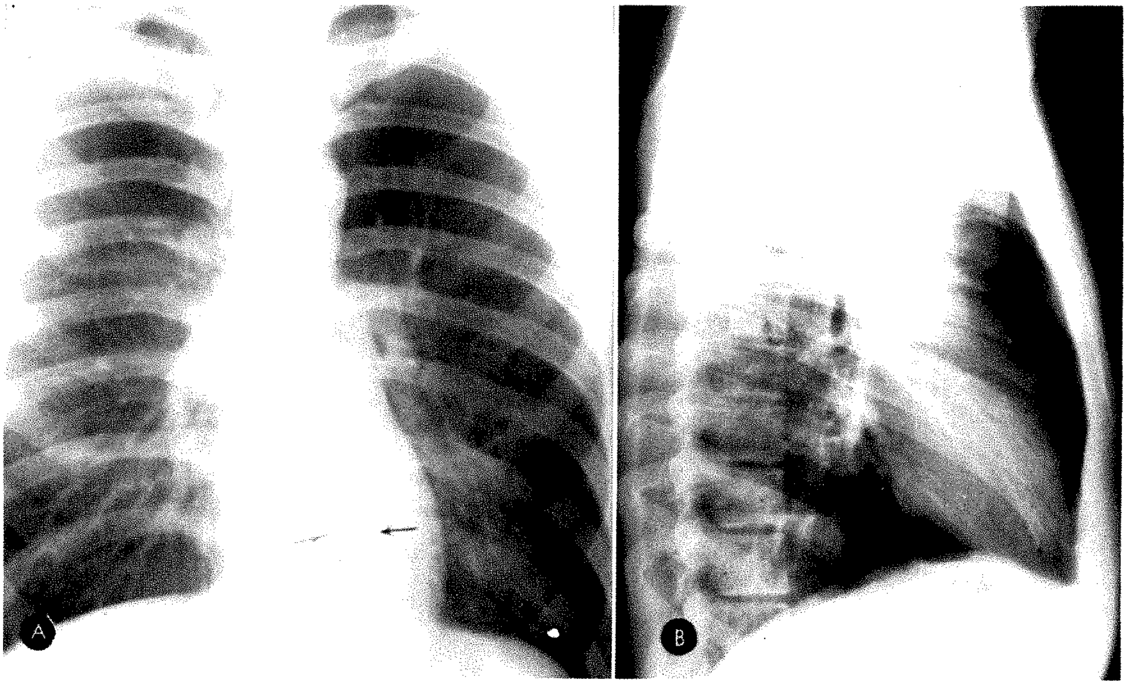

FIG. 8. Case III. (*A*) Posteroanterior roentgenogram of the chest, taken 10 hours after the patient fell from a 40 foot electric pole, shows a faint air fluid level located immediately to the left of the midline and projected over the heart shadow. The lung markings in the left base are accentuated, suggesting compression of the lung parenchyma by an adjacent expanding lesion such as a cyst. The mediastinum contains no observable free air. (*B*) A left lateral chest roentgenogram localized the air fluid level in the posterior basal segment of the left lower lobe.

positions, as well as across the table studies in the prone positions (not shown), established the dimensions of the air fluid containing cyst as 2×6×14 cm.

The patient was discharged from the hospital on December 31, 1964. A gastrointestinal study, performed on an outpatient basis on January 5, 1965, demonstrated the posterior basal segment, left lower lobe cyst to be independent of the gastrointestinal tract. The cyst no longer contained an air fluid level and had gotten smaller.

The patient has been reluctant to keep follow-up clinic appointments. The last chest roentgenographic study on March 3, 1965 (Fig. 9, *A* and *B*) demonstrated complete resolution of the large left lower lobe traumatic lung cyst. A small 2×1.5 cm., oval radiodensity believed to represent a residual pulmonary hematoma was present at the previous traumatic cyst site.

Comment. The history of vomitus containing supposedly swallowed blood in this case is closely akin to the hemoptysis of Case I. In both instances, the traumatic lung cyst originally contained fluid that disappeared on early subsequent roentgenograms. The acutely formed traumatic lung cyst present on the initial roentgenogram obtained just 10 hours post trauma was particularly large.

MECHANISM OF DEVELOPMENT

The basic event in the creation of a traumatic lung cyst, as well as many of the other above mentioned parenchymal injuries peculiar to nonpenetrating chest trauma, appears to be the transmission of a force from the external environment to the lung parenchyma.[8,20] The force may result from either direct or indirect trauma; direct trauma being that force applied primarily to the chest, whereas indirect trauma refers to the transmitted forces as in blast injuries, deceleration, or the secondary force exerted by the abdominal contents against the diaphragm.[20] In the present day setting, the trauma is most often ap-

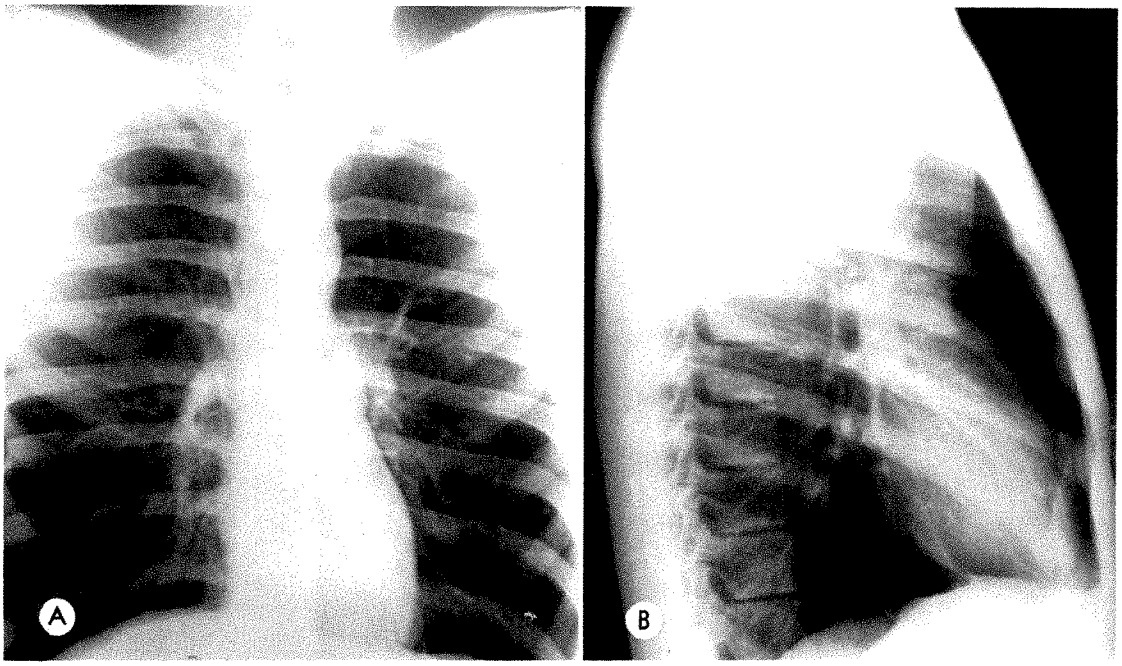

Fig. 9. Case iii. (*A*) Posteroanterior roentgenogram of the chest, taken 9 weeks post injury, demonstrates resolution of the retrocardiac lung cyst. A 1.5×2.5 cm., oval radiodensity, believed to be a pulmonary hematoma, is projected over the cardiophrenic angle. (*B*) A left lateral chest roentgenogram, taken at the same time, demonstrates no evidence of the previously present air fluid level. The traumatic air cyst has resolved.

plied directly to the thoracic wall as a result of rapid deceleration of the chest by some object located in the environment of the interior of an automobile. The semiflexible thoracic wall, particularly common to the child and young adult, is compressed, transmitting a force to the underlying, or in a contrecoup fashion (as in Case i) to the distant lung parenchyma.[9] As a result of the transmitted force a number of injuries can result. For example, in the case of the pulmonary contusion there is loss of vessel integrity so that there is perivascular, peribronchial, or intra-alveolar hemorrhage accounting for the bruised lung or pulmonary contusion. A pulmonary hematoma may be formed in a similar way.

Considering now the creation of an acute traumatic lung cyst, it is believed by the author that the transmitted force resulting from trauma suddenly compresses an area of lung, closing off a segment of the peripheral bronchial tree in such a fashion that a bursting explosive-like pressure is created within the lung lobule or area supplied by the occluded bronchus. Because escape of air through the upper air passage is insufficient, alveolar walls are successively ruptured until the confined pressure is spent. The walls of the resulting acute traumatic lung cyst are thus composed mainly of remnants of adjacent alveoli into which the air dissection has occurred.[3,9] The frequent association of an air fluid level with a traumatic lung cyst (Cases i, ii and iii) probably results from rupture of capillaries within the alveolar walls. If the entire cyst is filled with fluid, a blood cyst or pulmonary hematoma is formed.[2]

Another possible explanation as to the mechanism of development of an acute lung cyst comes from the writings of Williams and Bonte.[21] These authors describe the propagation of a concussion wave, initiated by blunt chest trauma, as creating shearing stresses capable of tearing the substance of the lung. As a result, an area of the lung parenchyma is torn, as opposed to being distended and ruptured, to explain the formation of a lung cyst—or in the terms of these authors—a pulmonary parencymal laceration. Regardless of how the injury is created, by bursting or tearing, its confinement to the lung parenchyma results in a traumatic lung cyst. If, on the other hand, the tear or rupture involves a pleural surface, a pneumothroax is produced.

The acute traumatic lung cyst, in the author's opinion, is to be distinguished from the cystic cavity which results from the resolution of a pulmonary hematoma. This latter type of cystic lung formation is described in the writings of Joynt and Jaffe,[8] Salyer, Blake and Forsee,[15] and Milne and Dick.[11] Undoubtedly, some of the lung cysts depicted in the articles by these authors acually represented acutely formed lung cysts. However, in several of the cases presented, the initial pulmonary lesion was manifested by a single solitary radiodensity not unlike a pulmonary hematoma or a localized pulmonary contusion. Concomitant with partial resolution of the density (blood clot), air fluid containing cavities became evident on the roentgenograms. Those cases treated by surgery involved cysts that were present for a long period of time (as long as 14 weeks). Upon histologic examination, these cysts were well defined structures with thick fibrotic walls measuring as much as 2.5 cm. and containing red-brown fluid or blood clots.[7,14] The author believes that these cystic lesions are secondary lesions; *i.e.*, cavities or pneumatoceles resulting from the resolution of a pulmonary hematoma or contusion, and differ from the acute traumatic lung cyst in their time and mode of development, and their initial roentgenographic appearance.

ROENTGENOGRAPHIC FINDINGS

The roentgenographic appearance of a traumatic lung cyst is variable, depending mainly on whether or not the cyst contains fluid or is associated with a pulmonary contusion.[8,17] Considering first the association of fluid, the cyst may be completely opacified and manifested by a single solitary oval "coin lesion-like" radiodensity within the lung field. The cystic nature of the lesion becomes apparent when the fluid (blood) begins to resolve or is coughed up, being expectorated or swallowed. More commonly, the lung cyst is represented by an isolated air fluid level in the lung parenchyma; the actual cyst wall being either imperceivable or quite thin. Still more commonly, the cyst wall may appear thick if there is a surrounding parenchymal reaction; this reaction is usually hemorrhagic in nature and represents contused lung parenchyma. If the associated pulmonary contusion is fairly extensive, the lung cyst may be partially or completely obscured. As the contusion resolves, usually in 3 to 6 days, the lung cyst, with or without an air fluid level, appears. As demonstrated in all 3 cases presented, the air fluid that may be associated with a traumatic lung cyst is often of a temporary nature, and may readily resolve or perhaps be evacuated by coughing.

Traumatic lung cysts are usually single and unilocular but it is not unusual to see more than one developing in an area of contused lung. They can be multilocular as in Case II and in the report by Greening *et al.*[5]

The size of the traumatic lung cysts is variable, ranging from small, 2 cm. in diameter, to large, 2×6×14 cm. (Case III). In instances where an actual cyst wall is not identifiable, the cyst size is indirectly obtained by measuring the length of the air fluid, if present.

A traumatic lung cyst can be located in any segment of either lung. The apices seem to be spared. It is important to realize that the cyst need not be confined to the immediate region of the chest wall injury. Trauma on the left may result in a parenchymal injury on the right (Case I). Injuries to the anterior chest not uncommonly result in parenchymal alterations posteriorly.

Traumatic lung cysts will practically always disappear in a period of weeks or months following their onset. The actual time interval is stated to be from 7 days to 3 months.[5] Since a large cyst may resolve quicker than a small cyst, it is reasonable to suspect that some of the cysts actually collapse (Case II) instead of resolving. Because traumatic lung cysts eventually disappear, observation is the main therapeutic measure unless the cyst becomes secondarily infected in which case surgery might be necessary.[5]

DIFFERENTIAL DIAGNOSIS

The differentiation of a traumatic lung cyst from a pulmonary abscess or a post-pneumonia pneumatocele is facilitated by a history of chest trauma and a negative history of any respiratory or systemic complaints compatible with an infectious process respectively. Tuberculous and mycotic cavity lesions may be differentiated from a traumatic lung cyst for the same reasons. Also, traumatic lung cysts spare the apices.

Pre-trauma comparative chest roentgenograms, when available, and a history of trauma will exclude the possibilities of congenital cysts and sequestrations. Then too, whereas these latter entities do not resolve, traumatic lung cysts eventually disappear; evidence that resolution is taking place can be present as early as 7 days post injury.

In the adult, a carcinoma of the lung may cavitate and present a thin-walled, air containing cystic structure not unlike a traumatic lung cyst. Here again, the history of the lesion in question is essential to the differentiation.

Other entitites peculiar to closed chest trauma such as pulmonary hematoma and localized areas of pulmonary contusion cannot, initially at least, be differentiated from a fluid filled traumatic lung cyst. The transient nature—3 to 6 days—of a pulmonary contusion distinguishes this entity from the longer lasting pulmonary hematoma and the fluid filled traumatic cyst. Since both of these latter two entities are associated with chest trauma and may pursue an identical clinical course in terms of treatment and duration, no significant diagnostic error is made if a fluid filled traumatic lung cyst is called a pulmonary hematoma or vice versa. It is again emphasized that most traumatic lung cysts are only partially filled with fluid and are manifested by an air fluid containing cyst within the lung parenchyma. These roentgenographic findings, when associated with chest trauma, should prompt one to diagnose a traumatic lung cyst.

SUMMARY

Three cases of acute lung cysts resulting from nonpenetrating chest trauma have been presented. This entity—termed traumatic lung cyst—has been discussed with reference to its mechanism of development, its roentgenographic features, and an appropriate differential diagnosis. Emphasis has been placed on the author's opinion that traumatic lung cysts represent still another common, acute, primary pulmonary lesion peculiar to closed chest trauma.

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THE ANGIOGRAPHIC FINDINGS IN FOUR CASES OF INFANTILE LOBAR EMPHYSEMA*

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LOBAR emphysema is an uncommon cause of respiratory distress in infancy. Although the diagnosis is usually established by plain roentgenograms of the chest and fluoroscopic observations, on occasion the diagnosis may be difficult. The purpose of this report is to present the pulmonary angiographic findings in 4 cases of infantile lobar emphysema.

REPORT OF CASES

CASE I. T.H., an 18 day old infant boy, was admitted with respiratory difficulty since birth. He was a 6 pound 14 ounce infant, the product of a full-term, normal pregnancy and delivery, who failed to have normal respirations for 15 to 20 minutes following birth. Increasing respiratory distress over the next 2 weeks with retractions, cyanosis out of oxygen tent, and noisy respirations led to his transfer to St. Louis Children's Hospital. On physical examination the pulse was 164 and the respiratory rate 100. Mild cyanosis was present. There were severe retractions and noisy breathing with prolonged and difficult expirations. Coarse rhonchi were heard in the chest. The remainder of the examination was normal.

Roentgenograms of the chest demonstrated right upper lobe emphysema with partial collapse of the right middle and lower lobe (Fig. 1, *A* and *B*). Multiple roentgenograms of the upper airway and an esophagogram were normal. The distal trachea was not well visualized. Fluoroscopy of the hemidiaphragms showed poor excursions but it was difficult to appreciate mediastinal shift. The heart on subsequent roentgenograms increased in size.

The patient developed a soft apical systolic murmur and hepatomegaly. There was electrocardiographic evidence of right ventricular hypertrophy. Laryngoscopy and bronchoscopy were normal except for questionable collapse of the right main stem bronchus. No abnormalities of the trachea were noted. A clinical diagnosis of coarctation of the aorta was made because of blood pressure discrepancy between the upper and lower extremities of 25 mm. Hg by the infratron method. Right heart catheterization revealed slight elevation of pressures in the right ventricle (35/10 mm. Hg) and main pulmonary artery (45/10 mm. Hg), but there was no evidence of left to right shunting.

Angiography demonstrated that the heart was displaced toward the left (Fig. 2, *A*, *B* and *C*). There was slight dilution effect in the pulmonary artery from a patent ductus arteriosus. The left main pulmonary artery doubled back on itself. The main right pulmonary artery was stretched and displaced to the left and inferiorly. The right upper lobe arteries were sparse, thin, and stretched. Peripheral vascular filling was poor. The middle and lower lobe vessels filled normally but were displaced downward and posteriorly. There was decreased size of the veins from the right upper lobe in addition to the displacement of the remaining veins draining the right lung.

A right upper lobe lobectomy was performed 2 days later. On entering the chest the right upper lobe was expanded and could not be collapsed. There appeared to be two small bronchi leading to the abnormal lobe. One had only a pinhole opening. The lobe could not be deflated after removal. Pathologically, no abnormalities of the mucosa or bronchial cartilage were seen. Postoperatively, the infant did poorly, with extremely difficult respirations, bradycardia, and cyanosis. Despite the usual medical measures, the infant died 12 hours following surgery at the age of 36 days.

At necropsy, the pertinent findings included moderately severe stenosis of the distal trachea at the bifurcation. The lungs showed dilated bronchi and intra-alveolar hemorrhage. A 1 mm. patent ductus arteriosus was found.

CASE II. S.S., a 2 day old male, was admitted with severe respiratory distress and cyanosis of 24 hours' duration. The pertinent physical

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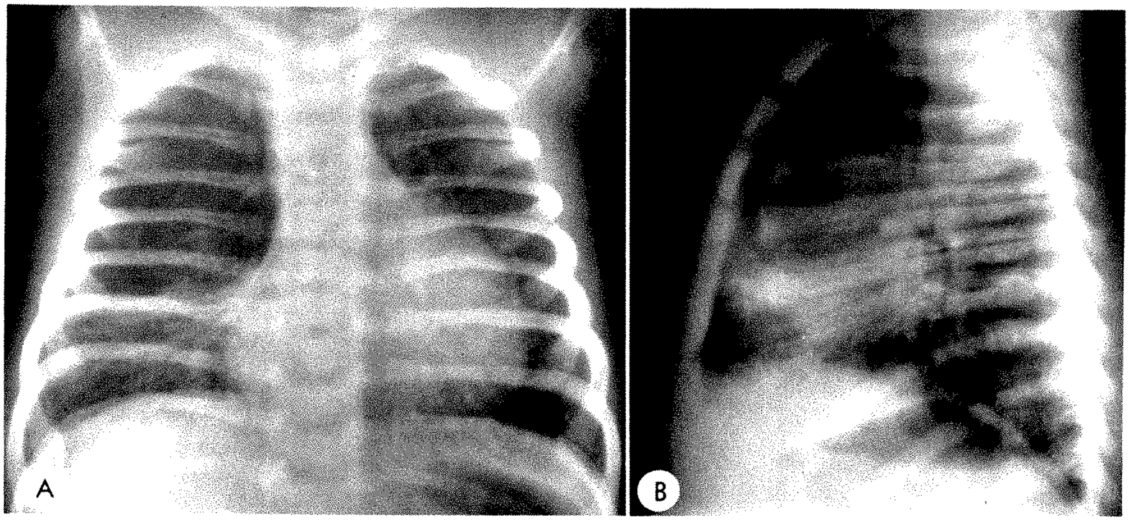

Fig. 1. Case 1. Posteroanterior (*A*) and lateral (*B*) roentgenograms of the chest show emphysema of the right upper lobe with compression of the remaining right lung and slight mediastinal shift.

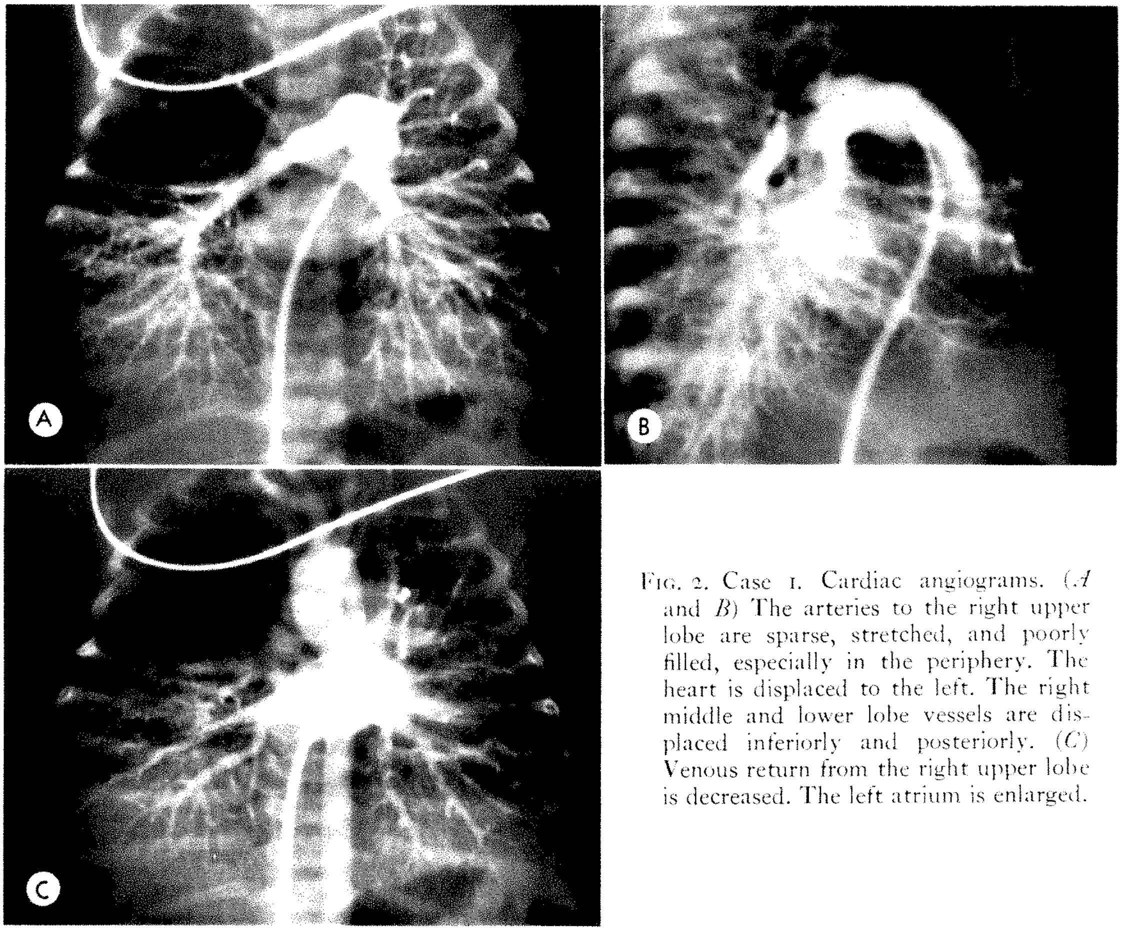

Fig. 2. Case 1. Cardiac angiograms. (*A* and *B*) The arteries to the right upper lobe are sparse, stretched, and poorly filled, especially in the periphery. The heart is displaced to the left. The right middle and lower lobe vessels are displaced inferiorly and posteriorly. (*C*) Venous return from the right upper lobe is decreased. The left atrium is enlarged.

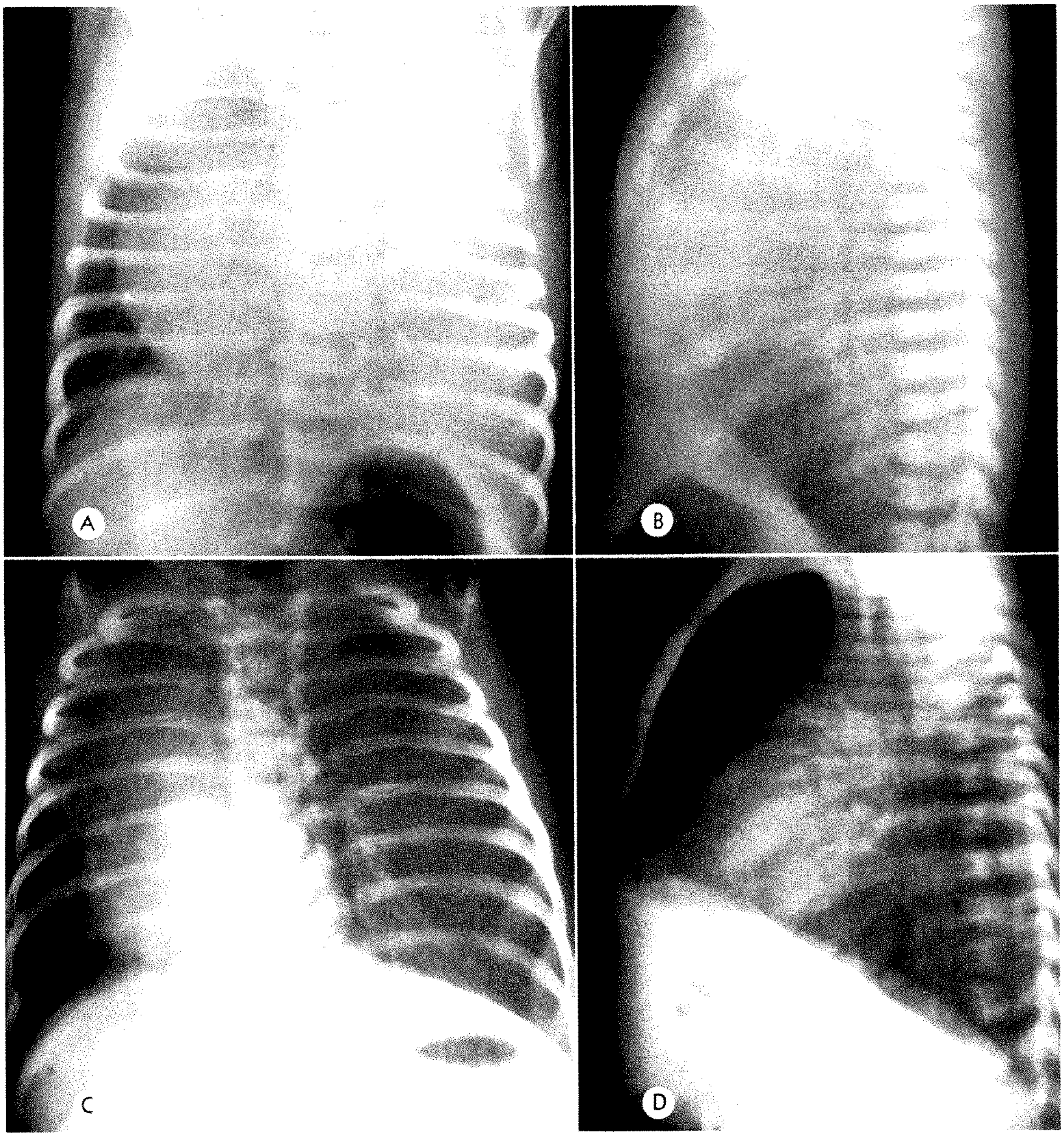

Fig. 3. Case ii. Posteroanterior and lateral roentgenograms of the chest. (*A* and *B*) Initial roentgenograms show extensive confluent infiltrates in an overexpanded left lung. (*C* and *D*) Seventeen days later an emphysematous left upper lobe is free of the infiltrates.

findings included cyanosis and decreased breath sounds over the left chest. There were no murmurs. An electrocardiogram was interpreted as showing right ventricular hypertrophy.

Chest roentgenograms showed a markedly expanded left lung containing confluent infiltration (Fig. 3, *A* and *B*). The mediastinum was shifted to the right. The infiltrates progressively cleared and a radiolucent lung remained (Fig. 3, *C* and *D*). Because the process was thought to involve the entire left lung, the roentgenographic interpretation was obstruction of the left main stem bronchus either from bronchomalacia or extrinsic pressure from aberrant vessels.

Right heart catheterization and angiocardiography were performed primarily to exclude an aberrant pulmonary vessel. Pressure and oxygen saturations in the right heart and pulmonary artery were normal. Angiography demon-

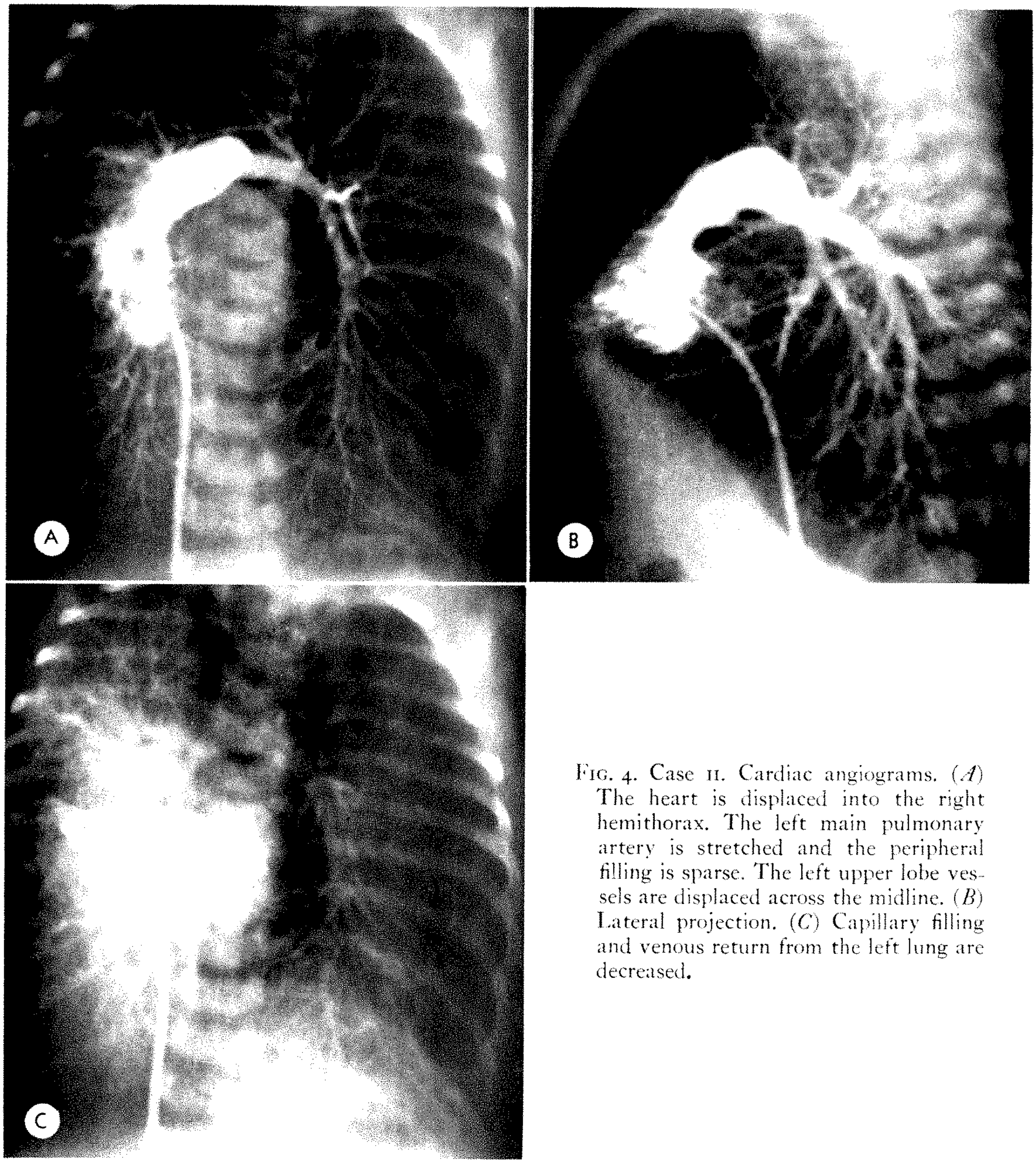

FIG. 4. Case II. Cardiac angiograms. (*A*) The heart is displaced into the right hemithorax. The left main pulmonary artery is stretched and the peripheral filling is sparse. The left upper lobe vessels are displaced across the midline. (*B*) Lateral projection. (*C*) Capillary filling and venous return from the left lung are decreased.

strated that the entire heart was displaced into the right hemithorax (Fig. 4, *A*, *B* and *C*). The right ventricle was slightly enlarged. The main pulmonary artery was markedly displaced toward the right. The left pulmonary artery was stretched. The right main pulmonary artery was oriented in a sagittal plane. The left upper lobe was emphysematous and its vessels were displaced across the midline into the superior portion of the right hemithorax. The vessels of the entire left lung were attenuated and lacked peripheral filling. The vessels of the right lung were crowded together and displaced posteriorly by the overexpanded left upper lobe and displacement of the heart into the right hemithorax. The capillary blush was not seen on the left. The veins from the left lung were smaller and more poorly filled than on the right. Reopacification of the pulmonary circulation secondary to a small patent ductus arteriosus was noted. The left heart and aorta were normal except for displacement.

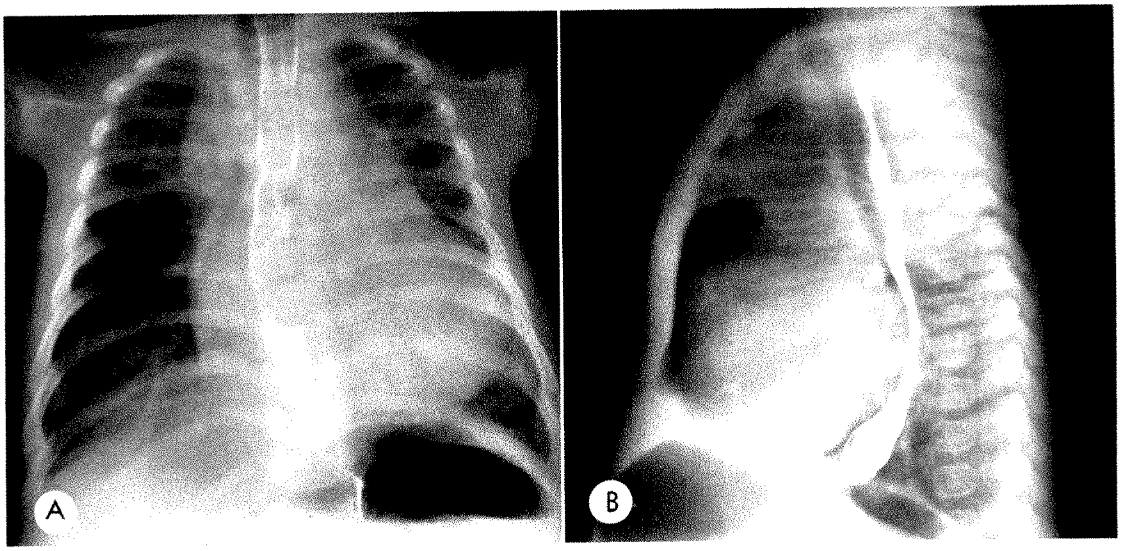

Fig. 5. Case iii. Posteroanterior (*A*) and lateral (*B*) roentgenograms of the chest show cardiomegaly with biventricular and left atrial enlargement and emphysema of the right middle lobe.

At the age of 3 weeks a thoracotomy was performed and a distended, nonelastic, cobblestone appearing left upper lobe was removed. The left upper lobe bronchus had the look and feel of a vein with no cartilage palpable. The superior segment of the left lower lobe also had a cobblestone appearance but was not overexpanded. It was not removed. The specimen did not collapse following removal of the bronchial clamps. The bronchus leading to the lobe was flaccid but not strictured. The postoperative course was uneventful. The surgical pathology diagnosis was lobar emphysema. In the year since his surgery, the patient has continued to have multiple bouts of pneumonia and upper respiratory tract infections, but these episodes have become less frequent and severe.

Case iii. A. H., a 2 month old infant girl, was admitted to St. Louis Children's Hospital because of rapid, shallow respirations and frequent vomiting since birth. There was a maternal history of rubella in the first trimester. The positive physical findings included weak femoral pulses, a discrepancy between the upper and lower extremity blood pressures, a Grade ii systolic murmur along the left sternal border, and an enlarged liver. An electrocardiogram was interpreted as showing right ventricular enlargement. A cardiac series showed an emphysematous right middle lobe and moderate cardiomegaly with left atrial and biventricular enlargement felt to be compatible with coarctation of the aorta and lobar emphysema (Fig. 5, *A* and *B*). Right heart catheterization revealed elevated right ventricular (46/0 mm. Hg) and main pulmonary artery (41–53/15 mm. Hg) pressures. No shunt was demonstrated. Angiocardiography demonstrated a right to left shunting through a patent ductus into the aorta. In the right middle lobe the arteries were stretched, smaller and lacked peripheral filling. The veins were also decreased in size. Venous return was to the left atrium which was enlarged along with the left ventricle. The aorta was wide to the area of a coarctation just superior to the origin of a patent ductus arteriosus.

Two days later a large patent ductus was ligated and transsected. The coarctation was excised and an end-to-end anastomosis carried out. The infant had an uncomplicated postoperative course. It is now only 2 months following the surgery and the patient is to be evaluated for possible removal of the right middle lobe.

Case iv. T.G., a 7 month old girl, was admitted for cardiac catheterization and angiography. The product of an uncomplicated pregnancy and delivery, the patient was discovered to have a right flank mass shortly after birth. Intravenous urography revealed no function of the right kidney. A tentative diagnosis of multicystic kidney was made and confirmed surgically. At 6 weeks of age, according to the

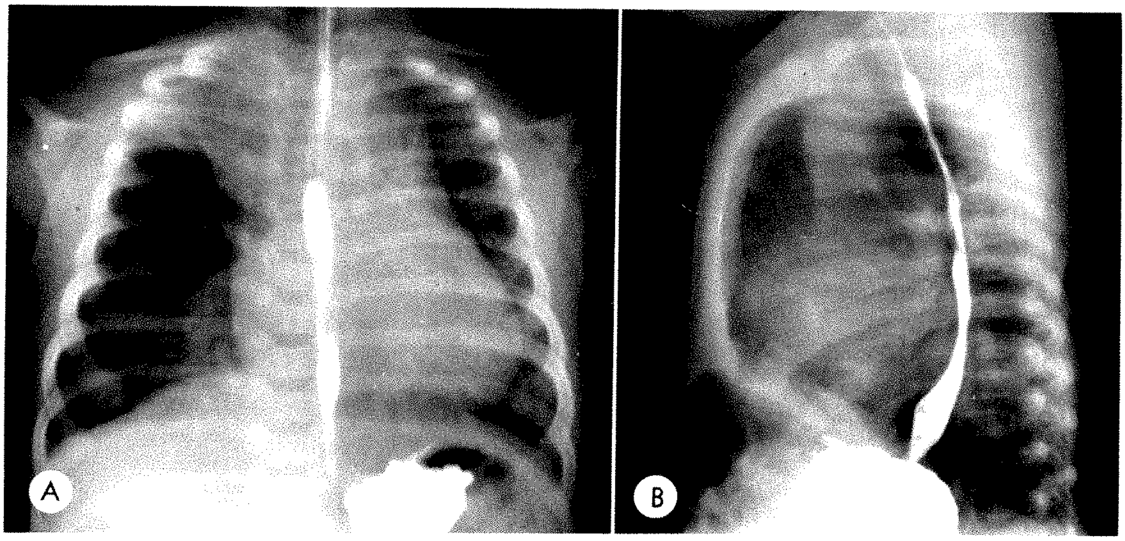

Fig. 6. Case iv. Posteroanterior (*A*) and lateral (*B*) roentgenograms of the chest demonstrate emphysema of the right middle lobe, partial collapse of the right upper and lower lobes, cardiomegaly, and slight overcirculation.

mother, the patient ceased breathing and had to be resuscitated by mouth to mouth respiration. The infant was then hospitalized with moderate respiratory distress. A few crepitant rales were heard in the left lung. A harsh Grade iv systolic murmur was heard along the left sternal border radiating over the entire precordium and into the back.

Chest roentgenograms showed emphysema of the right middle lobe with partial collapse of the right upper and lower lobes (Fig. 6, *A* and *B*). There was over-all increase in heart size with left atrial and right ventricular enlargement and overcirculation. The findings were compatible with a ventricular septal defect and congenital lobar emphysema of the right middle lobe. An electrocardiogram was interpreted as showing right ventricular enlargement. The patient quickly became asymptomatic.

On cardiac catheterization and angiography at the age of 7 months, a small left to right shunt was demonstrated at the ventricular level with normal pressure recordings. Angiography showed that the pulmonary artery and its branches were enlarged secondary to the left to right shunt. The vessels in the emphysematous right middle lobe were smaller and stretched. Peripheral vascular filling was poor in the involved lobe. Venous return was to the left atrium which along with the left ventricle was enlarged. The vessels in the remaining portion of the right lung were compressed. The right ventricle was re-opacified through the ventricular septal defect. No abnormal vessels were seen (Fig. 7, *A* and *B*).

Because the lobar emphysema is currently asymptomatic, the decision was made to treat the patient conservatively and consider surgical removal of the right middle lobe if pulmonary symptoms develop.

DISCUSSION

The symptomatology of infantile lobar emphysema is related to the altered pulmonary physiology and cardiac function from the overexpanded lobe. The involved lobe compresses the surrounding lung, displaces the mediastinum and can interfere with venous return to the heart. Approximately, one-half of the infants exhibit some respiratory distress in the first few days of life.[7] The major symptom of dyspnea may be of variable severity and progressive or intermittent. Recurrent respiratory infection may be the only manifestation. Cough, or lack of weight gain can be present. Stridor or wheezing, bilateral or limited to the same side as the lesion, may be heard. Cyanosis is seen with more severe alterations in pulmonary exchange. Fever is not generally present unless there is associated infection. The findings of diminished breath

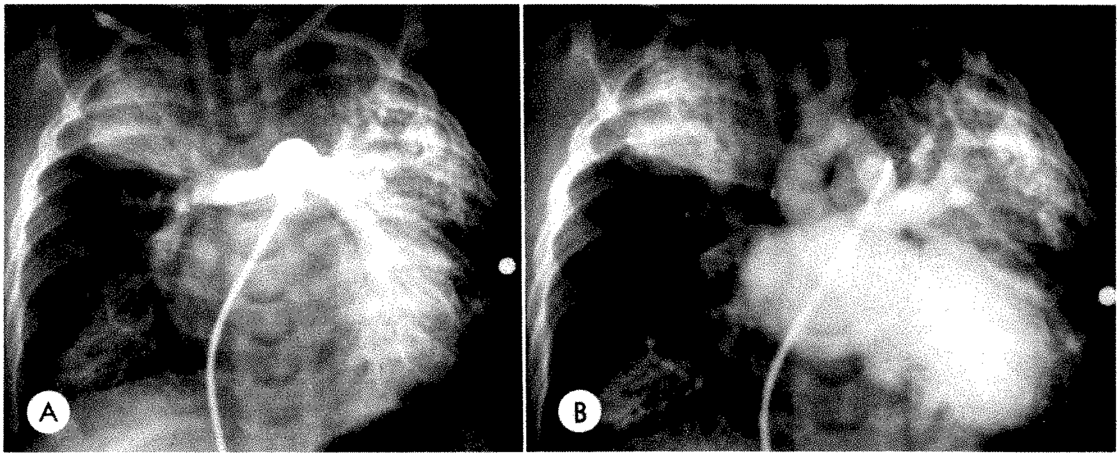

Fig. 7. Case iv. Cardiac angiograms. (*A* and *B*) The vessels in the emphysematous right middle lobe are sparse, smaller, and lack peripheral filling. The pulmonary artery and branches except in the diseased lobe are enlarged from left to right, shunting at the ventricular level. The left atrium and left ventricle are increased in size.

sounds, expansion of the involved hemithorax, and hyperresonance in an infant, either with or without cyanosis, would be suggestive of infantile lobar emphysema.

The basic pathology is some form of obstruction to the egress of air from the involved lobe. Postulated mechanisms include an abnormality or malformation of the bronchial cartilage, redundant intrabronchial mucosal folds or shelves acting as a check valve, compression of the bronchus by an abnormal vessel, or slight fibrosis of the alveolar walls rendering them unable to collapse during expiration.[1,4,5,7] The most common lobe involved is the left upper lobe. Bilobar involvement is unusual and the lower lobes are rarely affected. Microscopically, the process is relatively homogeneous. The alveoli are greatly dilated with some breakdown of their walls and coalescence into larger sacs. Bolande *et al.*[1] found slight fibrosis of the alveolar walls but others have not been able to confirm this. Congenital heart disease is seen in about 15 per cent of the cases.[7] Usually, no abnormality is seen by bronchoscopy but, occasionally, unusual flaccidity of the bronchial walls has been noted.

Chest roentgenograms usually establish the diagnosis and show an expanded radiolucent lobe through which vessels course.[2,6] The remaining lobe or lobes in the involved side will exhibit varying degrees of atelectasis and may be difficult to visualize. The mediastinum is shifted away from the emphysematous lobe and there is an excessive clear space behind the sternum in the lateral projection. The ipsilateral hemidiaphragm is depressed with restricted excursion. One of our patients had an expanded, somewhat opaque lung which cleared progressively into the typical overexpanded lobe (Fig. 3, *A–D*). Bronchography fails to fill the emphysematous lobe but will demonstrate the remaining compressed lobes.

Cardiac catheterization and angiography can be useful in selected cases, not only to help establish the diagnosis of infantile lobar emphysema but also to determine the anatomy in cases suspected of having an associated congenital cardiac defect. Angiography may establish the presence or absence of abnormal vessels which could partially obstruct the bronchus.

Angiographically, the pulmonary vessels of the affected lobes are stretched, attenuated, and spread apart throughout the emphysematous areas. Filling of peripheral vessels is sparse. Over-all arterial flow, including the affected lobe, is not appreciably slowed. The veins of the involved lobe are smaller. The vessels of the unaffected lobes adjacent to the involved lobe are displaced and compressed.

Differential diagnosis includes atelectasis of a portion of the lung with compensatory emphysema either of the remaining lung on that side or of the opposite lung. Agenesis or hypoplasia of a lung will result in mediastinal shift to the involved side. The above conditions can be separated by fluoroscopic observations or expiration roentgenograms. The emphysematous portions of the lung are able to deaerate normally during expiration. The mediastinum shifts away from the side of the atelectasis, agenesis or hypoplasia on expiration and shifts towards it on inspiration.

A pneumothorax or an air filled diaphragmatic hernia are distinguished from lobar emphysema by a lack of pulmonary vessels in the involved area. In addition, a pleural edge of the underlying compressed lung can be seen in a pneumothorax. A large Bochdalek hernia will usually contain multiple loops of bowel.

Congenital cystic adenomatoid malformations of the lung usually contain soft tissue structures such as immature lung in addition to air. In congenital cystic disease of the lung, no vessels course through the cyst and the outline of the cyst walls may be seen. A neurenteric cyst may be large and filled with air but is associated with vertebral anomalies in addition to a lack of vessels within the lesion. Foreign body aspiration is difficult to exclude but is usually seen in an older infant or child and there may be a history of aspiration. A foreign body produces more often obstructive emphysema to an entire lung.

Although most infants could be tided over the acute symptomatology by conservative management, infantile lobar emphysema, on occasion, can be fulminant requiring emergency lobectomy. Spontaneous remission has been reported.[3] Surgical resection of the involved lobe is the treatment of choice.[8]

SUMMARY

The findings on pulmonary angiography are reported in 4 cases of infantile lobar emphysema. These findings include attenuation, stretching, displacement, and sparse peripheral filling of the arteries in the affected lobe. The draining veins are smaller. The vessels in the adjacent lobes are displaced and compressed.

Angiography can be useful in these cases where the diagnosis is in doubt.

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THE ROLE OF THE BARIUM SWALLOW EXAMINATION IN EVALUATION OF PEDIATRIC PNEUMONIAS*

By OLGA M. BAGHDASSARIAN GATEWOOD, M.D., *and* JOHN J. VANHOUTTE, M.D.†
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RECURRENT or persistent pneumonia in infancy and childhood presents a difficult problem in diagnosis and management. Entry of ingested material into the respiratory tree is often overlooked as a causative mechanism. Barium swallow examinations with fluoroscopic observation and cinefluorography, if available, are essential in the evaluation of conditions that may cause aspiration. Therefore, this examination is performed routinely in our institution on any infant or child in whom persistent or recurrent pneumonic infiltrations present a clinical problem.

The conditions that may be demonstrable by barium swallow studies are:

- I. Defective swallowing mechanism
  - 1. Micrognathia
  - 2. Riley-Day syndrome
  - 3. Brain damage and neurologic deficit
  - 4. Idiopathic incoordination of the newborn
- II. Direct tracheo-esophageal communication.
  - 1. Congenital H-type tracheo-esophageal fistula
  - 2. Postoperative recurrent fistulae
  - 3. Congenitally deficient posterior laryngeal wall
- III. Esophageal obstructions or dyskinesia resulting in overflow aspiration
  - 1. Congenital and acquired esophageal stenoses
  - 2. Achalasia
  - 3. Vascular rings
  - 4. Disturbed motility in postoperative congenital esophageal atresia.

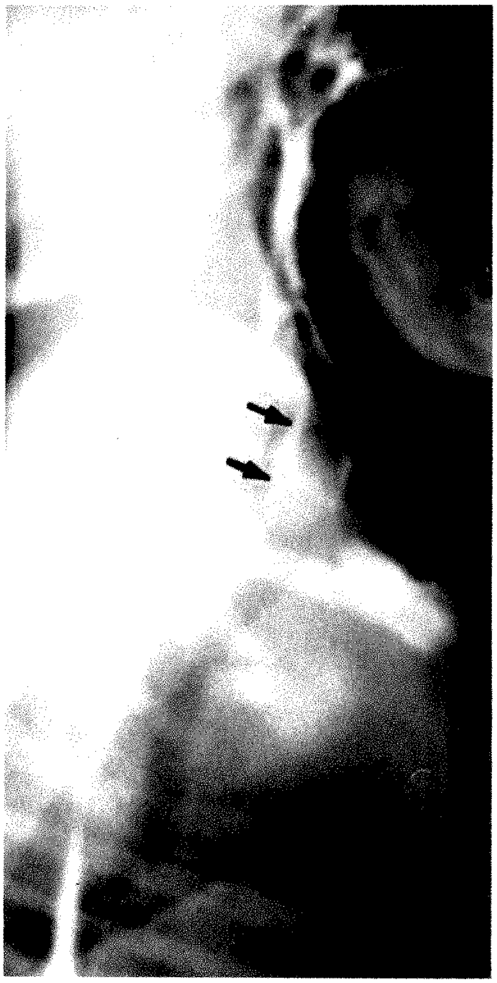

Fig. 1. Spot roentgenogram of an 8 day old infant with micrognathia, showing spillage of barium into the trachea (arrows).

I. DEFECTIVE SWALLOWING MECHANISM

Incoordination of the swallowing mechanism resulting in aspiration of contrast material at the hypopharyngolaryngeal

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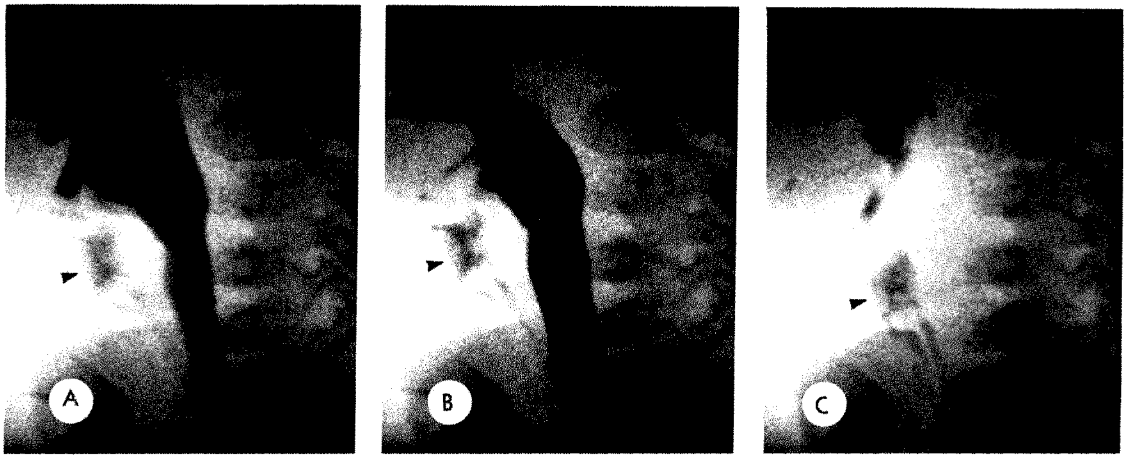

FIG. 2. (*A*, *B* and *C*) Aspiration of barium into the larynx and trachea (arrows) demonstrated in a 5 year old boy with familial dysautonomia (selected frames from cine-esophagram).

level is frequently seen in association with micrognathia as well as in patients with Riley-Day syndrome.[2,4]

Micrognathia, as the name indicates, is a congenital deformity consisting of an abnormally small mandible. It is a feature of the Pierre-Robin syndrome when glossoptosis and cleft palate are associated.[7] The infant with micrognathia feeds with difficulty and may develop repeated aspiration pneumonia. Contrast examination of the swallowing mechanism shows spillage of the opaque material into the trachea at the laryngeal level (Fig. 1). The opaque material may also be regurgitated into the nasopharynx and then aspirated.

The Riley-Day syndrome, characterized by defective lacrimation, excessive perspi-

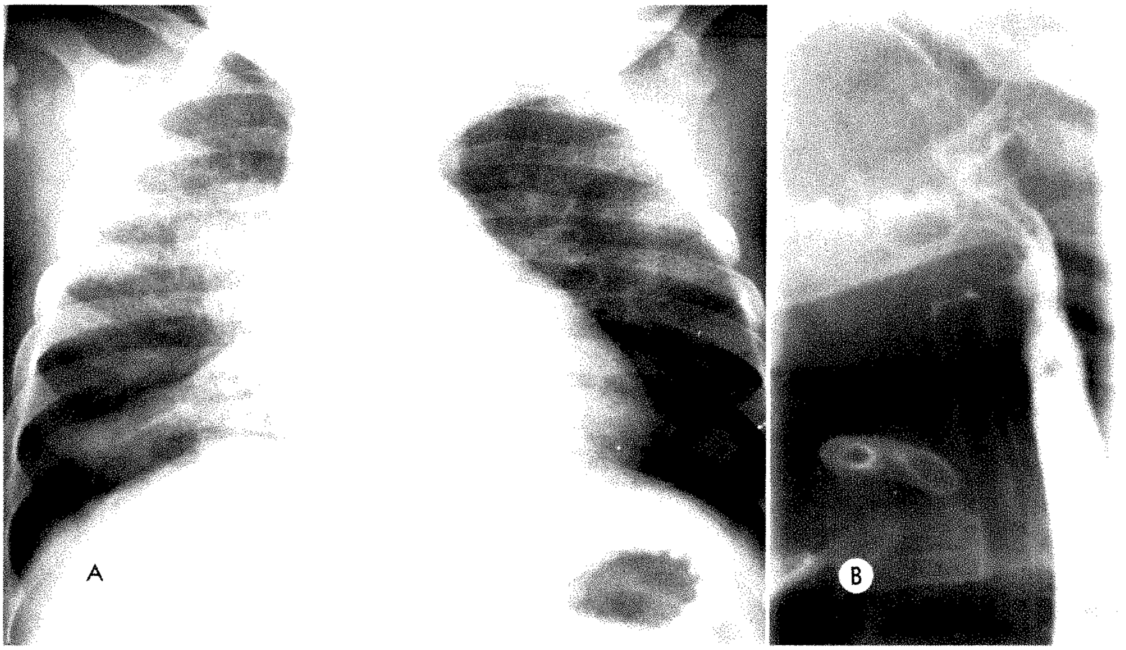

FIG. 3. (*A*) Anteroposterior chest roentgenogram of a 6 month old retarded, flaccid female infant demonstrates central consolidations involving the dependent (posterior) portions of the lungs. Note loss of volume of the right lower lobe. (*B*) Spot roentgenogram showing spillage of barium into the trachea.

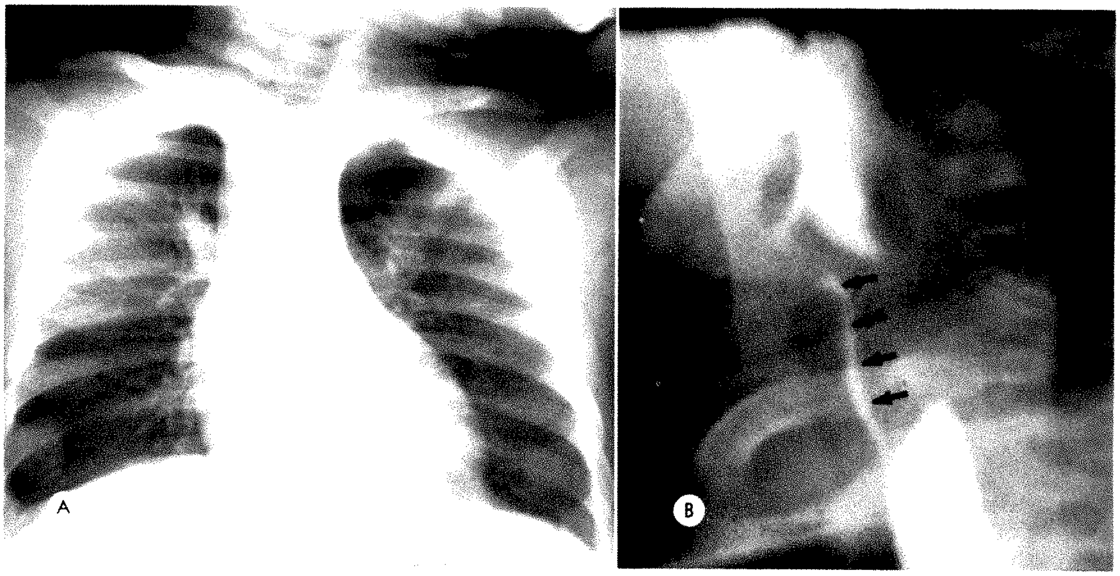

Fig. 4. (*A*) Anteroposterior roentgenogram of the chest of a 4 month old retarded infant demonstrating bilateral central conglomerate infiltrations with peripheral hyperaeration. (*B*) Spot roentgenogram showing spillage of contrast material into the trachea outlining its posterior wall (arrows).

ration, inconstant hypertension, vomiting and emotional lability, is also associated with recurrent pulmonary infections. Cineroentgenograms of the esophagus demonstrate overflow of the contrast material into the larynx and trachea during swallowing (Fig. 2, *A*, *B* and *C*). The amount of fluid aspirated usually increases with progressive swallowing, suggesting that fatigue of the deglutition function may play a part. The aspirated contrast material does not induce coughing and the patient is usually unaware of its occurrence. Abnormal motor function of the esophagus has also been observed in this condition.[2] Marked clinical improvement has resulted in one patient in whom limitation of the volume of fluid ingested at any one time and elimination of all fluids other than clear water from the dietary regimen were undertaken.

Occasionally, infants with brain damage or nonspecific neurologic deficit may develop widespread bronchopneumonia and demonstrate aspiration on examination (Fig. 3, *A* and *B*; and 4, *A* and *B*). Incoordination of the swallowing mechanism is also seen in neuromyopathies (Fig. 5, *A-D*).

Pharyngeal dyskinesia resulting in aspiration also may be encountered in the newborn.[1] This temporary functional abnormality may be severe enough to mimic an H-type tracheo-esophageal fistula clinically. Occasionally, a temporary gastrostomy may be required to prevent further aspiration.[5] The cause of this condition is not known but it probably is related to immaturity (Fig. 6).

II. DIRECT TRACHEO-ESOPHAGEAL COMMUNICATIONS

Extensive and recurrent bronchopneumonic infiltrations in infants and children should always suggest the possibility of an H-type tracheo-esophageal fistula. This type of fistula may occur as a congenital malformation or may result as a postoperative complication of a repaired esophageal atresia and tracheo-esophageal fistula. A very striking associated feature is the marked distention of the stomach and bowel which results from the free flow of air from the trachea into the gastrointestinal tract (Fig. 7, *A-D*). Roentgenographic demonstration of these fistulae is not consistent because the tracheo-esophageal communication is not always patent.[2] Repeated ex-

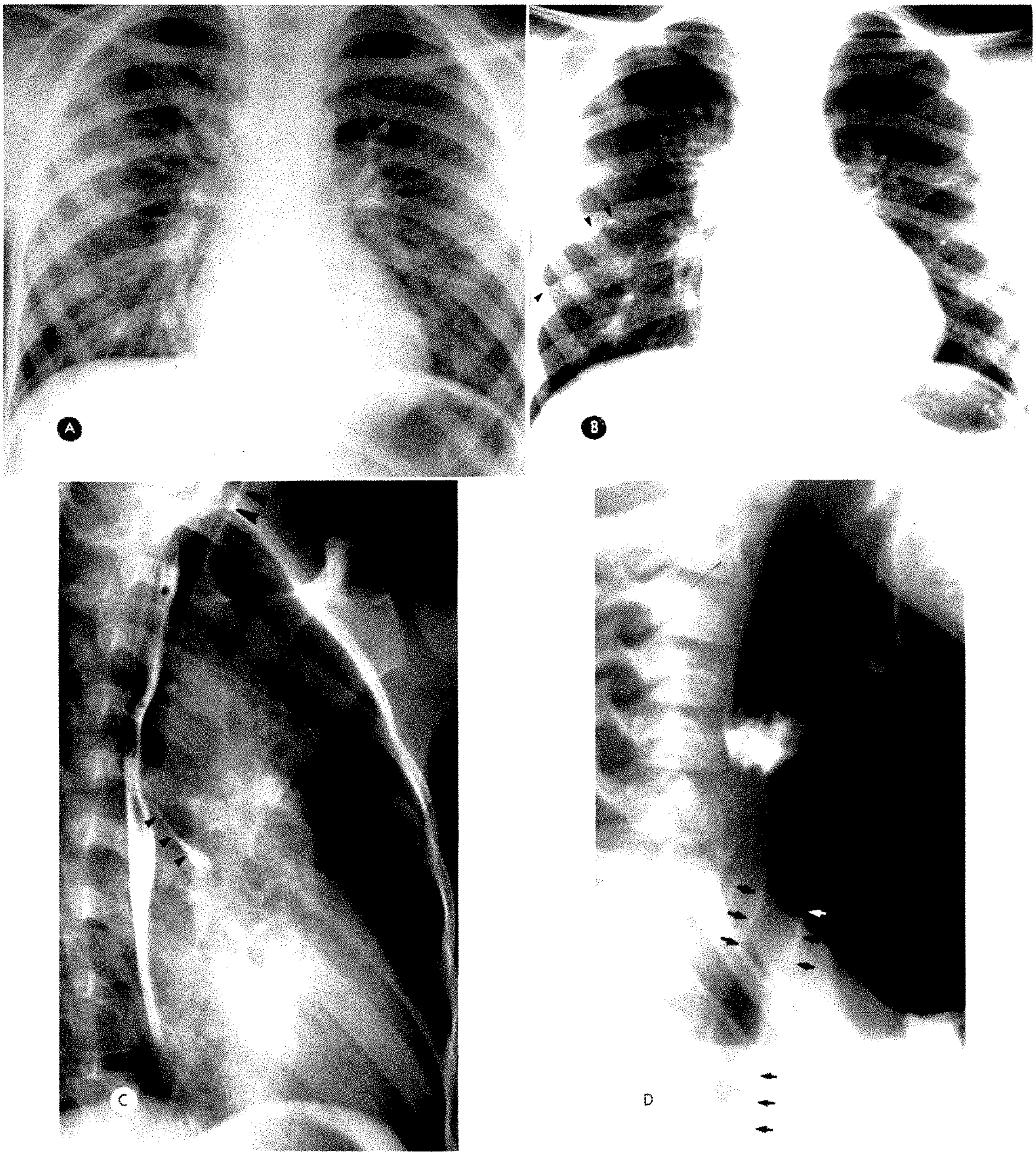

FIG. 5. (*A*) Posteroanterior roentgenogram of the chest in a 5 year old boy with an undiagnosed neuromyopathy. Note the extensive central streaky infiltrates. (*B*) Posteroanterior roentgenogram of the chest obtained following thin barium swallow shows contrast material in the peripheral bronchioles (arrows). (*C*) Right anterior oblique chest roentgenogram obtained following creamy barium ingestion shows entry of the barium into the tracheobronchial tree (arrows). (*D*) Spot roentgenogram showing pooling of the thin barium suspension in the valleculae and pyriform sinuses and overflow into the trachea (arrows). No reflux into the nasopharynx occurred.

aminations are necessary, if the diagnosis is strongly suspected. The infant should be placed in the prone oblique position since these fistulae are directed anteriorly and usually superiorly. Many consecutive barium swallows must be observed since the fistula may only be fleetingly demonstrated.

Infants with persistent or recurrent pneumonia after repair of esophageal atresia and tracheo-esophageal fistula should be re-examined. Postoperative recurrences of tracheo-esophageal fistulae are usually found at the site of the anastomosis.[7]

Congenital absence of deficiency of the posterior laryngeal wall is a very rare anatomic abnormality which may simulate both clinically and roentgenographically pharyngeal incoordination or a high H-type tracheo-esophageal fistula.[9]

III. ESOPHAGEAL OBSTRUCTIONS OR DYSKINESIA RESULTING IN OVERFLOW ASPIRATION

Esophageal obstructions are an infrequent cause of recurrent pneumonia. They include congenital esophageal stenosis, acquired strictures, vascular rings, and achalasia. These obstructions cause dilatation of the esophagus, retention of food and fluid, and subsequent overflow spillage of esophageal contents into the larynx (Fig. 8, *A*, *B* and *C*). Achalasia is seldom encountered in infancy and childhood.[4]

Following repair of esophageal atresia and tracheo-esophageal fistula, persistent and recurrent pneumonias in infants and children more often result from abnormal motility in the esophagus than from recurrent tracheo-esophageal fistula or stenosis.[3,6] The lack of peristalsis in the distal segment of the esophagus prevents free emptying of the esophagus. When sufficiently distended, the distal segment responds with a spastic contraction. Since there is no orderly progression of peristalsis, this mass contraction expresses the contrast agent in both cephalic and caudal directions. The retrograde propulsion may be severe enough to produce overflow aspiration.

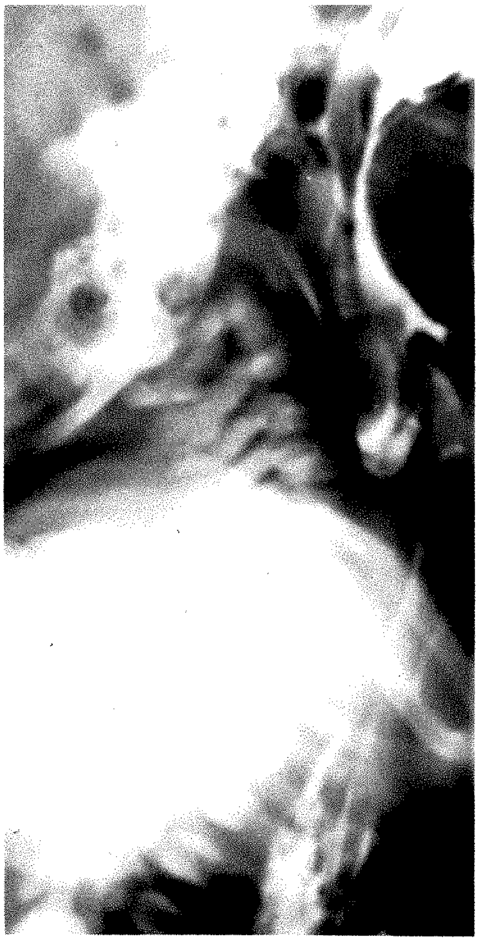

Fig. 6. Aspiration of contrast material in a 1 day old infant with pharyngeal incoordination; the infant was thought to have a tracheo-esophageal fistula clinically.

SUMMARY

The barium swallow study is an important examination in the evaluation of unexplained chronic and recurrent pneumonias in infancy and childhood. Hypopharyngeal and esophageal abnormalities causing aspiration and demonstrable by the barium swallow examination are enumerated and discussed.

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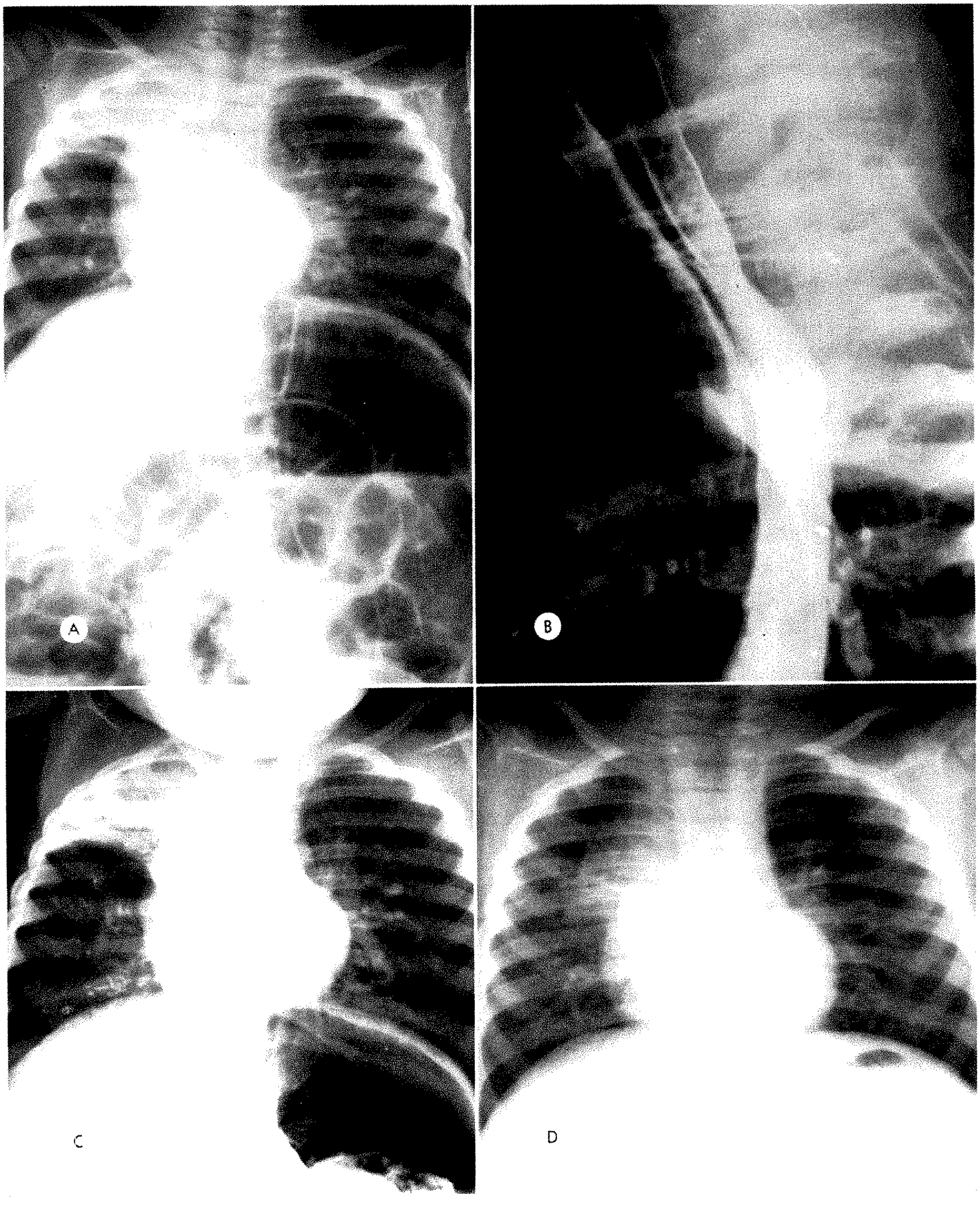

Fig. 7. (*A*) Anteroposterior roentgenogram of the chest and upper abdomen in a 1 year old boy with an H-type tracheo-esophageal fistula. Extensive bilateral infiltrations, right upper and lower lobe atelectasis and marked gaseous bowel distention are seen. (*B*) Spot roentgenogram demonstrating the fistula. (*C*) Anteroposterior roentgenogram obtained following the barium swallow showing contrast material in the bronchi. (*D*) Anteroposterior chest roentgenogram obtained 1 year postoperatively showing an essentially normal appearance. Note complete re-expansion of the previously atelectatic lobes and increase in the size of the thymus.

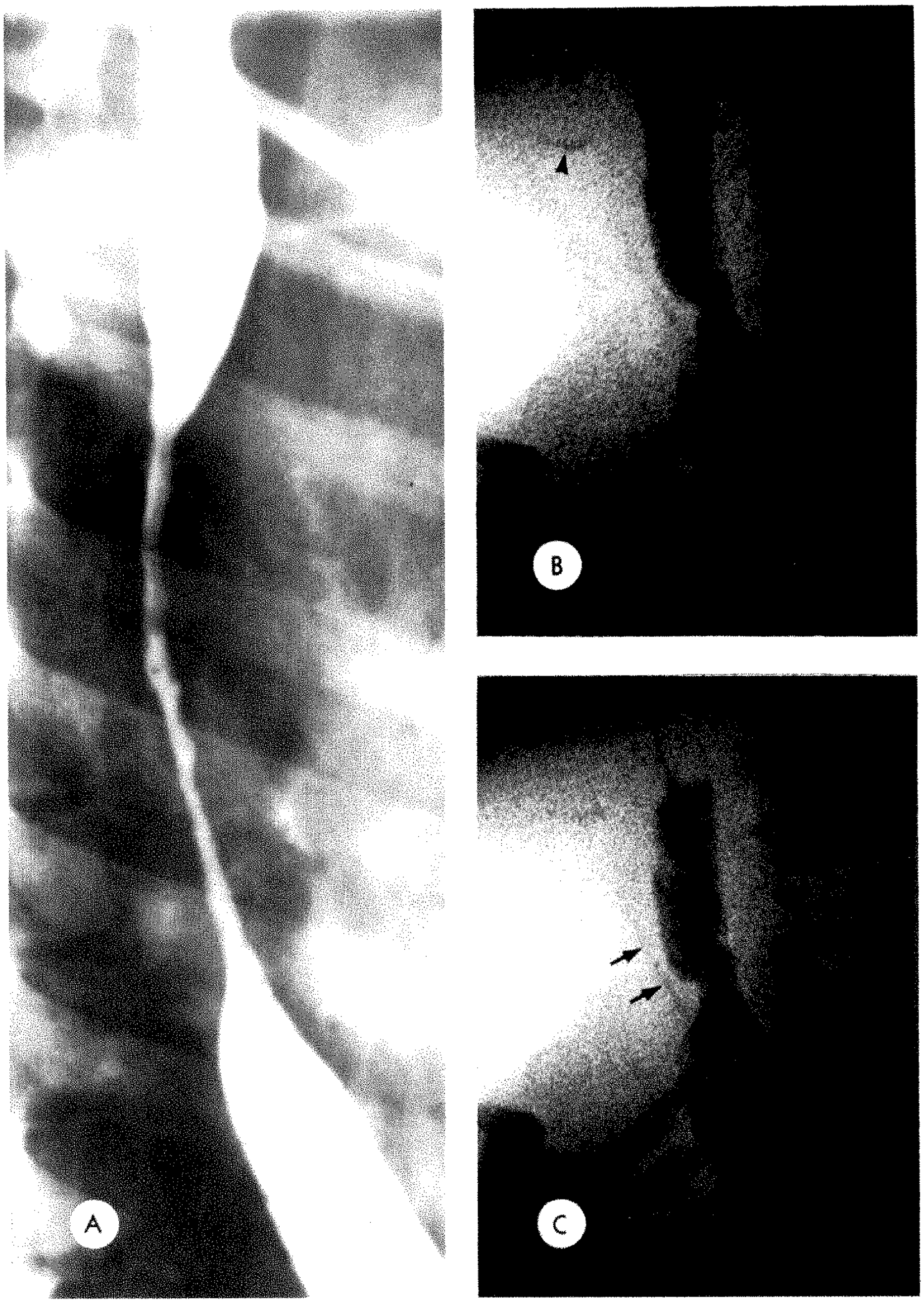

Fig. 8. (*A*) Roentgenogram following barium swallow in an 18 month old boy with caustic esophageal stricture. (*B* and *C*) Cineroentgenograms demonstrate spillage of opaque material into the trachea (arrows).

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PERSISTENT ESOPHAGOTRACHEA

THE MOST SEVERE DEGREE OF LARYNGOTRACHEO-ESOPHAGEAL CLEFT

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NATURE'S arrangements for keeping food out of the air passages and an excess of air out of the alimentary tract are complicated but generally satisfactory. The disasters that arise with malfunction of this separation of the two streams—most notably aspiration pneumonia—are fairly well known. Should a condition arise where separation of food from air is impossible, the organism would inevitably succumb. Such a condition is persistent esophagotrachea,[17] in which the esophagus and trachea are a single tube, united for most or all of the length of the trachea. This congenital anomaly, exemplified by the following case, is the most severe form of laryngotracheo-esophageal cleft,[2] the mild forms of which involve the larynx alone.

REPORT OF A CASE

This 36 hour old girl (CHMC 57-15-86) was admitted with exstrophy of the bladder. The pregnancy had been uneventful. Difficulty with oral secretions was noted soon after birth, and respiratory distress and episodes of cyanosis developed. There had been one small sugar-water feeding.

Examination showed a lethargic dyspneic baby with considerable oral and pharyngeal mucus. She made no sound when crying, but at the time no specific significance was attached to this. Intercostal retractions were present, and there were scattered rhonchi.

A chest roentgenogram (Fig. 1) showed collapse of the right upper lobe, hyperinflation elsewhere, and scattered shadows consistent in appearance and distribution with aspiration.[11] Fluoroscopy showed that an opaque tube could readily be passed into the stomach. Contrast material given by mouth simultaneously filled both the trachea and the esophagus, which were intimately associated; their shadows could not be satisfactorily separated. The impression from the fluoroscopy and spot roentgenograms (Fig. 2, *A* and *B*) was that there probably was a high unusually large H-type tracheo-esophageal fistula.

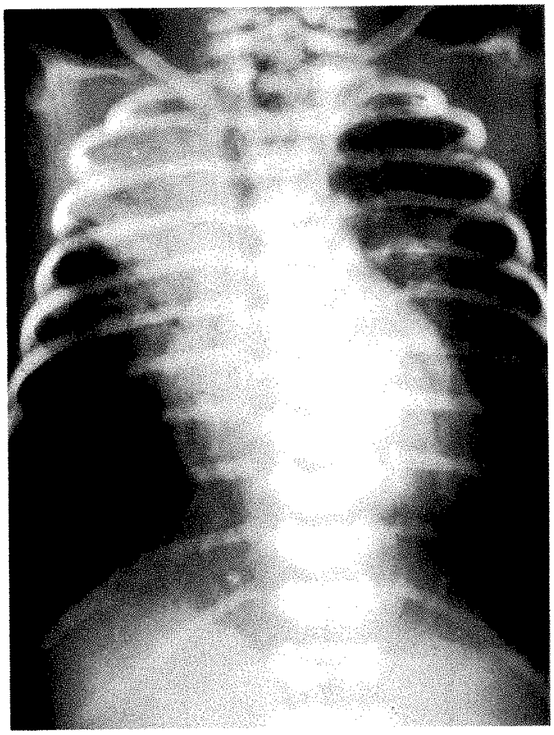

Fig. 1. Chest roentgenogram on admission, at the age of 36 hours. There is obvious consolidation and loss of volume of the right upper lobe. Less marked changes are seen on the left. Hyperinflation is evident elsewhere. There is an unusual lucency in the superior mediastinum, presumably air within the esophagotrachea.

During anesthetization and intubation, the posterior larynx was noted to be abnormal. Surgical exploration showed that the trachea and esophagus were in communication from the pharynx to within 1 cm. of the carina, with a cleft-like fissure joining one lumen to the other (Fig. 3, *D* and *E*). The common tracheo-esophageal tube was transsected, and the upper

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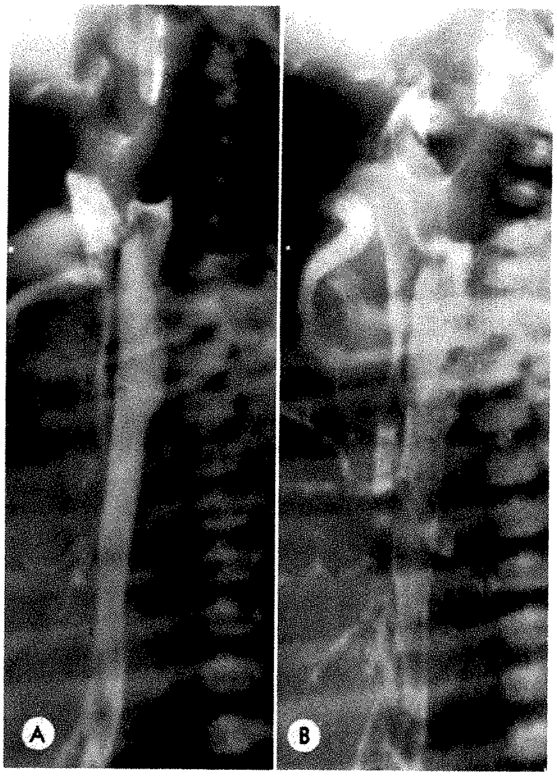

Fig. 2. (*A* and *B*) Fluoroscopic lateral spot roentgenograms. There is an opaque sump tube in the pharynx. Opaque medium was given by bottle. The roentgenograms show immediate and simultaneous filling of trachea and esophagus. The shadows of the trachea are separated, although incompletely, presumably by the unfused lateral infoldings (see Fig. 3*E*). No single fistulous tract was ever visible, nor was clear-cut transglottic aspiration shown. The trachea is somewhat small, and its outline and that of the esophagus are slightly irregular. The soft tissue density at the top of the esophagus may represent muscle spasm.

stump was marsupialized to the skin of the neck. A back wall of esophageal tissue was constructed for the lower trachea, and this was brought to the skin as a tracheostomy. The lower esophageal remnant was closed.

Postoperatively, the child's respirations became progressively more labored and irregular, and she died at 3 days of age.

Autopsy showed severe bladder exstrophy, rectal and vaginal stenosis, and only one umbilical artery. The spleen was deeply lobulated and looked like a bunch of grapes. The heart and great vessels were normal. There was widespread pneumonia, with abscesses, from which *Pseudomonas aeruginosa* was cultured. The presence of persistent esophagotrachea was confirmed. The operative repair was intact.

DISCUSSION

Uncommon as persistent esophagotrachea is, its embryogenesis is fairly easy to understand. The respiratory tract is first

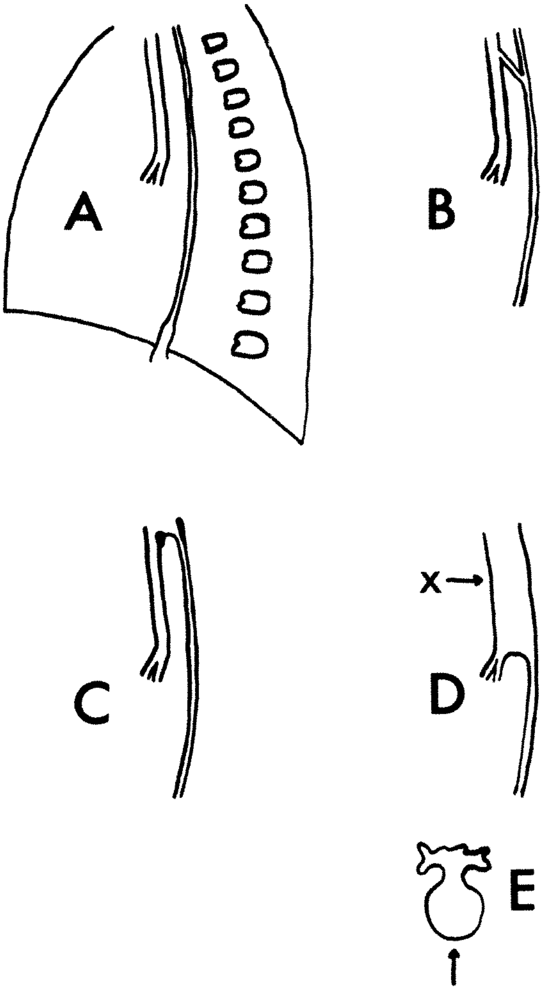

Fig. 3. (*A*) Diagram of normal esophagus and trachea as seen from the side. (*B*) Isolated high H-type tracheo-esophageal fistula. (*C*) Short laryngotracheo-esophageal cleft; the airway and the gullet are in continuity down into the upper trachea. (*D*) Persistent esophagotrachea, the most severe degree of laryngotracheo-esophageal cleft, with total continuity down to the carina. (*E*) A diagrammatic cross-section of the common tube at the level of "X," showing that the lateral infoldings approach each other but have not fused.

recognizable as a pouch from the anterior aspect of the primitive foregut.[3] With elongation of the gut, the pouch also progresses caudad, but it becomes separated from the forming esophagus by the union, from below upward, of infoldings of tissue from each side of the common tube. These folds form the back wall of the trachea and larynx and the front wall of the esophagus and hypopharynx. The alimentary and respiratory tracts normally are thus separated by these folds up to the top of the larynx. The rudimentary trachea repeatedly divides at its lower end to form bronchi, bronchioles, and alveoli, while the esophagus continues to elongate.

Incomplete development and nonfusion of the lateral folds is a reasonable explanation for the anomalous persistence of the common esophagotracheal channel in the case described above. An H-type tracheo-esophageal fistula presumably represents local nonfusion of these folds. Deviation of the folds towards or to the posterior wall of the esophagus would explain congenital esophageal stenosis or atresia, and origin of the infoldings from the posterior wall of the lower part of the common tube could account for a distal tracheo-esophageal fistula. Presumably, incompleteness, dorsal or ventral displacement, or twisting of these lateral infoldings accounts not only for the usual types of esophageal atresia and tracheo-esophageal fistula but also for such very rare anomalies as origin of the trachea or of one of the bronchi from the lower esophagus.[3,8]

Reports of at least 18 cases of some dedegree of laryngotracheo-esophageal cleft can be found in the literature, and there are allusions[5,6,10] to a few others. About half of the defects were reported to be short[2,4–6,9,12,13]—laryngeal only, or extending down to the topmost tracheal rings—but in the other cases (as in the infant presented above) the nonfusion extended to or almost to the carina.[1,7,15–17] The first report[14] (without surgical or autopsy confirmation, apart from digital examination of the throat through the mouth) appeared in 1792. Two of the cases of short laryngotracheo-esophageal cleft were in sisters[5,6] and 2 of the infants with virtually total esophagotracheal communication were siblings.[17] Three cases of atresia of the esophageal portion of the common tube[17] or of esophageal atresia below a short cleft[4] have been reported, and these infants have also had lower tracheo-esophageal or tracheogastric fistulas.

Like newborns with esophageal atresia with or without tracheoesophageal fistula, patients with persistent esophagotrachea (the nonfusion extending to or almost to the carina) early accumulate and have difficulty ridding themselves of mucus in the mouth and pharynx. As mucus or feedings enter the lungs, dyspnea and cyanosis develop. A gastric tube can be successfully passed unless other anomalies are present, however, in contrast with the ordinary case of esophageal atresia. Aspiration is earlier, more severe, and less likely to be limited to feeding time than in the usual isolated H-type fistula, which is seldom diagnosed in the first few days of life. Many and perhaps all of these babies, because of the posterior defects in their larynxes, have no voice.

Endoscopy performed by one who is alert to the condition will show that the larynx is splayed posteriorly and that the trachea and esophagus are in communication, especially free and gaping during inspiration. In some of the reported cases, however, endoscopy failed to reveal the lesion.

Chest roentgenograms of patients with persistent esophagotrachea demonstrate pneumonia, often severe, of the aspiration type,[11] and the shadows of the air-containing trachea and esophagus may be abnormal. Fluoroscopy during swallowing will show that the opaque material enters the esophagus and trachea more or less simultaneously, as in massive transglottic aspiration or an unusually large prompt-filling tracheo-esophageal fistula. (If the medium is administered via a tube in the middle third of the esophagus, simple functional aspiration can probably be excluded.) The shadows of the esophagus and trachea will be difficult or impossible to separate. No single fistulous tract between the two will be demonstrable. Careful analysis of cinefluorography of swallowing may sometimes be helpful. A definite diagnosis probably cannot be made by roentgenography alone, although these roentgenographic findings in a newborn with secretion difficulty, aspiration pneumonia, aphonia, and tube-patent esophagus should strongly suggest a severe form of laryngotracheo-esophageal cleft.

In a few cases of persistent esophagotrachea or shorter cleft, the correct diagnosis has been reached and repair attempted. The most hopeful report is that of Pettersson;[12] his patient's laryngotracheo-esophageal cleft was high and short and was successfully repaired, even to restoration of normal voice. (Incomplete information reaching the writer verbally indicates that a second successful repair has been carried out elsewhere, on a voiceless baby whose laryngotracheo-esophageal cleft presumably was similarly short; the correct diagnosis was made endoscopically, after a barium swallow examination had suggested an H-type fistula.) In at least 2 cases, cervicothoracic exploration (undertaken with a diagnosis of isolated tracheo-esophageal fistula) failed to detect either a fistula or the actually present short cleft; the lower ends of the clefts were mistaken for normal laryngopharyngeal junctions. Except as mentioned above, all cases have been fatal in early infancy. The more extensive the communication between airway and gullet, the freer will be the aspiration, and the sooner the death of the infant may be expected.

SUMMARY

A case of a newborn with persistent esophagotrachea, a condition in which the respiratory and alimentary tracts are in continuity down to the carina, is reported. Persistent esophagotrachea is the most severe degree of laryngotracheo-esophageal cleft. Infants with persistent esophagotrachea have difficulty with secretions from birth and promptly develop severe aspiration pneumonia (as in cases of esophageal atresia). Usually, a tube can be advanced readily into the stomach however (as in cases of H-type tracheo-esophageal fistula without esophageal atresia). Aphonia is common, possibly universal. Fluoroscopy will show immediate entry of ingested contrast material into the trachea; the shadows of the esophagus and trachea will be difficult or impossible to separate from each other, but no isolated fistula will be demonstrable. Endoscopy should be able to confirm the lack of fusion of the posterior walls of the larynx and trachea. Neither roentgenologic evaluation, nor endoscopy, nor surgical exploration has heretofore been uniformly successful in reaching the correct diagnosis however; this must be at least partly due to lack of knowledge of the condition.

Because diagnosis has been delayed or inaccurate or both and because the problems of surgical repair are formidable, persistent esophagotrachea of the degree exemplified by the case reported here has until now been a rapidly lethal malformation, although 2 cases of milder degrees of laryngotracheo-esophageal cleft have apparently been successfully repaired.

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BILATERAL CONGENITAL EVENTRATION OF THE DIAPHRAGM

CASE REPORT WITH ROENTGEN MANIFESTATIONS

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AVNET[1] recently described for the first time 2 extensively studied cases of bilateral congenital eventration of the diaphragm. The rarity of this condition prompted us to report a patient seen in our hospital.

REPORT OF A CASE

History. The patient, a 5 week old infant who was found to have a heart murmur, was referred to the radiology department for a cardiac series. The mother had noted nothing unusual about the child except for an infrequent cough. Development was normal.

Past History. Delivery was by spontaneous breech. The birth weight was 5¼ pounds. There was no history of first trimester illness or medication.

Family History. No history of congenital anomalies was obtained.

Physical Examination. The patient was a well formed, 5 week old infant in no distress. No cyanosis, abnormal respiratory rate, or chest retraction were seen. Breath sounds were normal. A Grade II/VI pansystolic murmur was heard throughout the chest, but best along the left parasternal border. Liver and spleen were not palpable.

Course. The left sided eventration was repaired on August 28, 1964 and the right side on October 16, 1964. The second operation was followed by a stormy postoperative course but the patient improved rapidly and has been well since. Excellent results were obtained by surgery.

ROENTGENOGRAPHIC FINDINGS

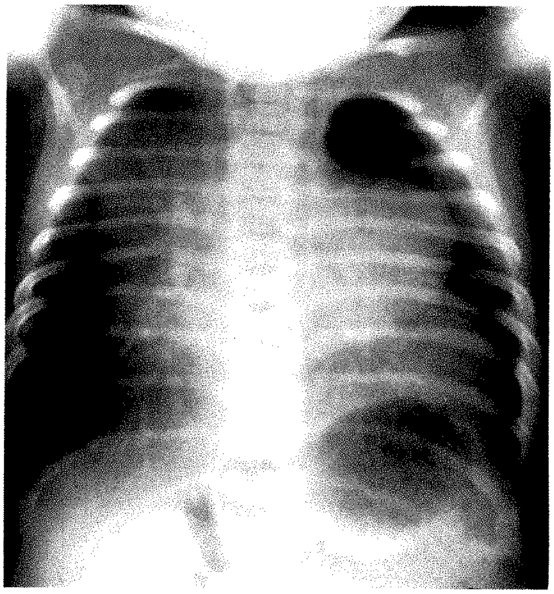

FIG. 1. Initial chest roentgenogram showing bilateral asymmetric widening of the mediastinum and upward displacement of the apex of the heart.

The initial chest roentgenogram (Fig. 1) showed a bilateral asymmetric widening of the mediastinum by a soft tissue mass. The heart was displaced upward and to the left so that the apex was in the fourth posterior interspace. The mass was felt to be located anteriorly as there was no unusual density in the posterior half of the chest in the lateral roentgenogram. At fluoroscopy, this soft tissue mass appeared to move up and down somewhat with the stomach, suggesting that it was closely related to the diaphragm, if not a part of it. After an oral administration of barium, no evidence of herniation of the abdominal viscera into the thoracic cavity was seen. The initial impression was that the subcardiac mass arose in the region of the diaphragm either from the mediastinum or from the diaphragm itself. Whether the mass on the right was a part of an abnormal structure or a normal but unusually enlarged thymus was difficult to ascertain. To reach a more definite diagnosis, 30 cc. of air was injected into the peritoneal cavity. The pneumoperitoneum roentgenograms (Fig. 2 and 3) demonstrated eventration of both leaves of the diaphragm.

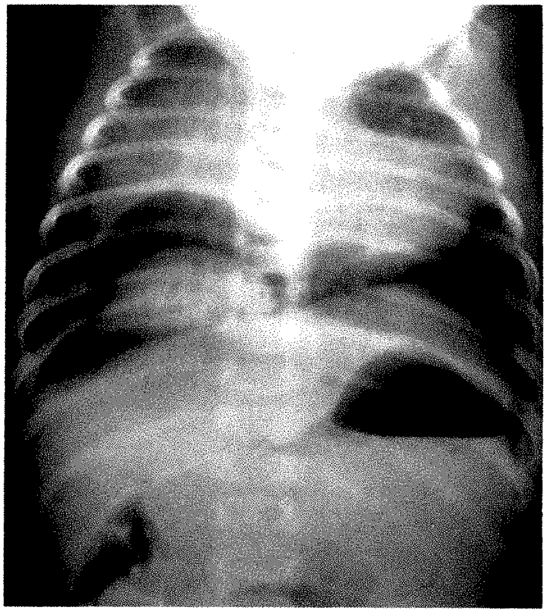

Fig. 2. Pneumoperitoneum showing eventration of both leaves of the diaphragm.

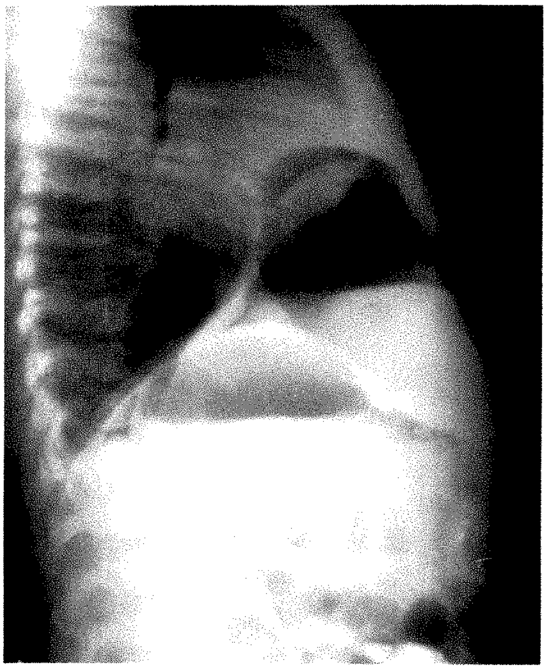

Fig. 3. Lateral pneumoperitoneum study.

DISCUSSION

At surgery, defects were found in the muscular portion of the diaphragm anteriorly and laterally. These were covered only by peritoneum and pleura. The liver was firmly attached to the under surface of the right leaf by ligamentous structures. There was no herniation of any portion of the abdominal contents into the chest cavity. These findings define diaphragmatic eventration. Displacement of the heart upward and to the left may be a valuable roentgenologic sign if one has a high enough level of suspicion upon seeing an unusual silhouette on the posteroanterior and lateral roentgenograms of the chest. This unusual displacement is the result of the right sided eventration which will not permit the usual displacement of the heart seen when the condition is unilateral. A coincidental finding in our study was the absence of an obvious splenic shadow below the diaphragm on the left side. This, too, may suggest a congenital anomaly insomuch as splenic agenesis is seen associated with one or more other congenital abnormalities and particularly eventration of the diaphragm. Chromosomal studies were not done.

SUMMARY

A third case of bilateral congenital diaphragmatic eventration is presented. The condition may be asymptomatic. The displacement of the heart upward and to the left is a valuable sign.

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GASTRIC LEIOMYOMA IN A CHILD[*]

REPORT OF A CASE

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LEIOMYOMAS represent only 1 per cent of all gastrointestinal neoplasms reported[5] and of these slightly more than half are gastric in origin.[4] A recent review of the world literature by Skandalakis and associates[6] disclosed 1,017 cases of both benign and malignant smooth muscle tumors of the stomach since Morgagni reported the first case in 1762. Eighty-four per cent were between the ages of 30 and 70 years, and this age incidence is confirmed by 3 timely reports from Aberdeen,[2] Indianapolis,[1] and Atlanta.[7] Uncommon as they are in adults, leiomyomas of the stomach are even rarer in children and most physicians will gain little experience in their diagnosis or treatment.

The most common symptoms and findings are gastrointestinal hemorrhage with or without anemia, pain, and a palpable mass. The clinical presentation is far from characteristic, however, and of 17 cases of benign leiomyoma in one review,[7] only 2 were correctly diagnosed prior to surgery and only 3 included it in the differential diagnosis.

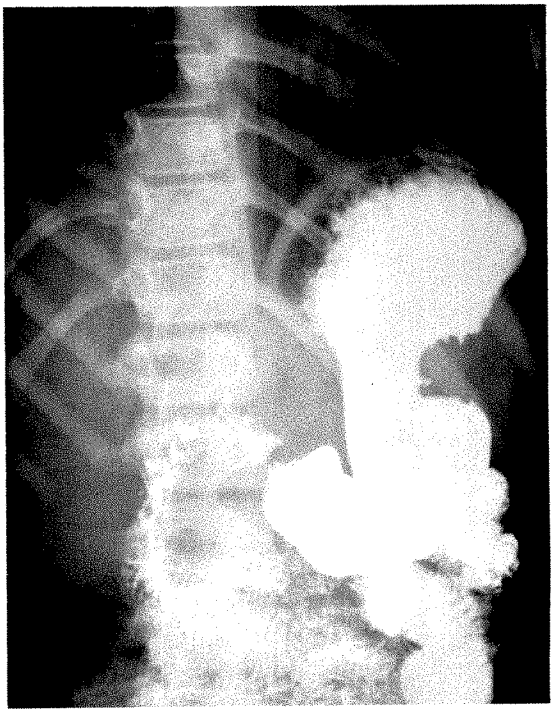

FIG. 1. The leiomyoma is outlined on the greater curvature. There is a central ulcer niche and a normal rugal pattern covering the remaining surface of the neoplasm.

These tumors arise from the smooth muscle wall, muscularis mucosae, and, rarely, the blood vessels in the gastric wall.[6] If they grow toward the serosa, few or no roentgenographic findings will be present. However, if they remain intramural or grow toward the lumen, a rounded filling defect in the barium filled stomach will often be seen. As demonstrated in the case report to follow, an ulcer niche on the dome and a nearly normal rugal pattern covering the mass are frequent findings. Peristalsis is usually normal. Twenty years experience at the University of Maryland Hospital revealed that the radiologist was more often correct in the preoperative diagnosis than the referring physician.[3]

While these tumors are well circumscribed grossly, they rarely have an intact capsule when examined under the microscope. The difference between a leiomyoma and a leiomyosarcoma is determined by the amount of cellular activity.[6]

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This material has been reviewed by the Office of The Surgeon General, Department of the Army, and there is no objection to its presentation and/or publication. This review does not imply any indorsement of the opinions advanced or any recommendation of such products as may be named.

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REPORT OF A CASE

An 11 year old well developed, well nourished Caucasian male enjoyed good health until late August, 1964, when he complained of substernal pain following vigorous exercise. He experienced several similar episodes during the next few days and when examined prior to admission, both tachycardia and pallor were present. A hematocrit at this time was 21 per cent.

On September 8, 1964, he was admitted to Letterman General Hospital. There was no history of diarrhea, constipation, melena, or bloody stools. Physical examination revealed no palpable masses in the abdomen. His pulse rate was 116 per minute, hemoglobin was 6.0 gm. per cent, hematocrit was 20 per cent and the reticulocyte count was 6.6 per cent. There was marked hypochromia and microcytosis of the red blood cells. Laboratory examination of the stools demonstrated the presence of occult blood.

Proctoscopic examination was negative and a barium enema study was interpreted as negative in spite of large quantities of fecal material in the colon. When the remainder of the gastrointestinal tract was studied with barium, a 5×2.5 cm. intraluminal mass with central ulceration was found on the greater curvature of the stomach (Fig. 1). The gastric mucosa covering the tumor appeared normal in profile except in the area of ulceration. When the barium enema roentgenograms were reviewed, the neoplasm was seen outlined by air within the stomach (Fig. 2). This retrospective observation emphasizes the importance of simple studies in the diagnosis of gastrointestinal disease when complete and careful attention is given to all areas of the roentgenogram.

The patient's anemia was corrected by transfusions of packed red blood cells and whole blood, prior to a sleeve gastric resection. Microscopic examination of the specimen showed the tumor to be a leiomyoma. He has made an uneventful recovery.

SUMMARY

A case of a gastric leiomyoma in an 11 year old boy is reported. The symptoms, signs, and roentgenographic findings are reviewed. It is hoped that cognizance of these will make a correct preoperative diagnosis likely.

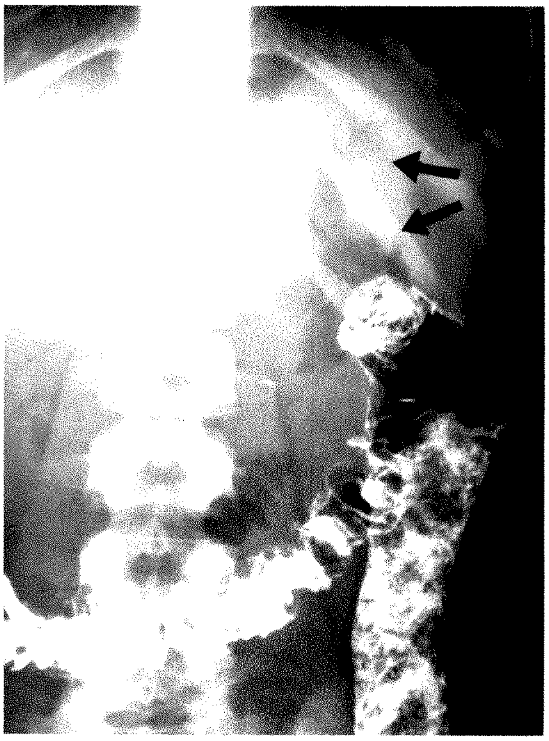

Fig. 2. A portion of the postevacuation roentgenogram shows the tumor outlined by gastric air. The arrows point to the base of the mass.

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PORTAL VEIN GAS*

A CASE REPORT WITH SURVIVAL

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GAS in the portal venous system has been described as a grave prognostic sign foreboding death, without exception. In a recent paper,[5] the first survival was reported in a 48 year old male with chronic ulcerative colitis who developed pneumoperitoneum and portal vein gas following a double-contrast barium enema examination. The following case is the first infant with gas in the portal vein known to have lived.

REPORT OF A CASE

A full-term, normally delivered white female infant, 13 days old, was admitted because of fever, poor appetite, and swelling with resistance to motion of her right thigh. Osteomyelitis of the right femur was suspected and intramuscular penicillin was administered although roentgenograms were negative. After several days of therapy, the signs of osteomyelitis disappeared but diarrhea and a markedly distended abdomen appeared. Roentgen examination, 4 days after admission, showed right upper lobe pneumonia and distention of bowel compatible with paralytic ileus. The pneumonia slowly resolved after 2 weeks therapy with intramuscular penicillin, kanamycin, and streptomycin, but poor appetite, diarrhea and abdominal distention persisted. Roentgen examination, 14 days after admission, revealed marked distention of the small and large bowel, intramural intestinal gas, and gas in the portal venous system (Fig. 1, *A*, *B* and *C*). Enterococci were cultured from blood, stools and urine. Antibiotics were continued and the intramural and portal vein gas disappeared, although distention persisted. Because of failure to improve and a falling hemoglobin, the baby was transferred to Babies Hospital in New York City, 27 days after admission with a diagnosis of osteomyelitis and necrotizing enterocolitis.

At Babies Hospital, pathogenic *E. coli* was cultured from stool specimens, which became culturally negative on oral polymixin, although no demonstrable improvement occurred. Osteomyelitis of the right femoral neck became apparent on roentgenograms and she was given intravenous staphcillin and chloromycetin. The patient's appearance improved, she gained weight, and her white blood cell count fell. She was discharged 51 days after admission. Subsequent examination by her pediatrician has revealed a healthy baby without sequelae.

DISCUSSION

Gas in the portal venous system has been previously reported in 31 patients,[1–12] of which 13 have been infants. In the adults, the most common precipitating cause was mesenteric vascular occlusion with bowel necrosis. It has also been described secondary to acute hemorrhagic pancreatitis with extreme bowel dilatation, closed loop obstruction with intestinal gangrene, necrotizing enterocolitis, bowel necrosis with severe atherosclerosis of mesenteric vessels without occlusion, and in a 35 year old diabetic in acidosis and shock who had marked bowel distention and congestion without vascular obstruction or necrosis.

In children, portal vein gas has been seen in the following: necrotizing enterocolitis in premature infants; intestinal obstruction with peritonitis in imperforate anus and duodenal atresia; esophageal atresia and peritonitis following surgery; idiopathic diarrhea; mesenteric vein thrombosis with gangrene of bowel and severe dehydration; and in erythroblastosis fetalis.

The origin of the gas in the portal vein is undecided. One theory suggests that gas under tension in distended bowel escapes into the mesenteric veins and portal vein through a bowel wall altered by impaired circulation due to the severe distention or

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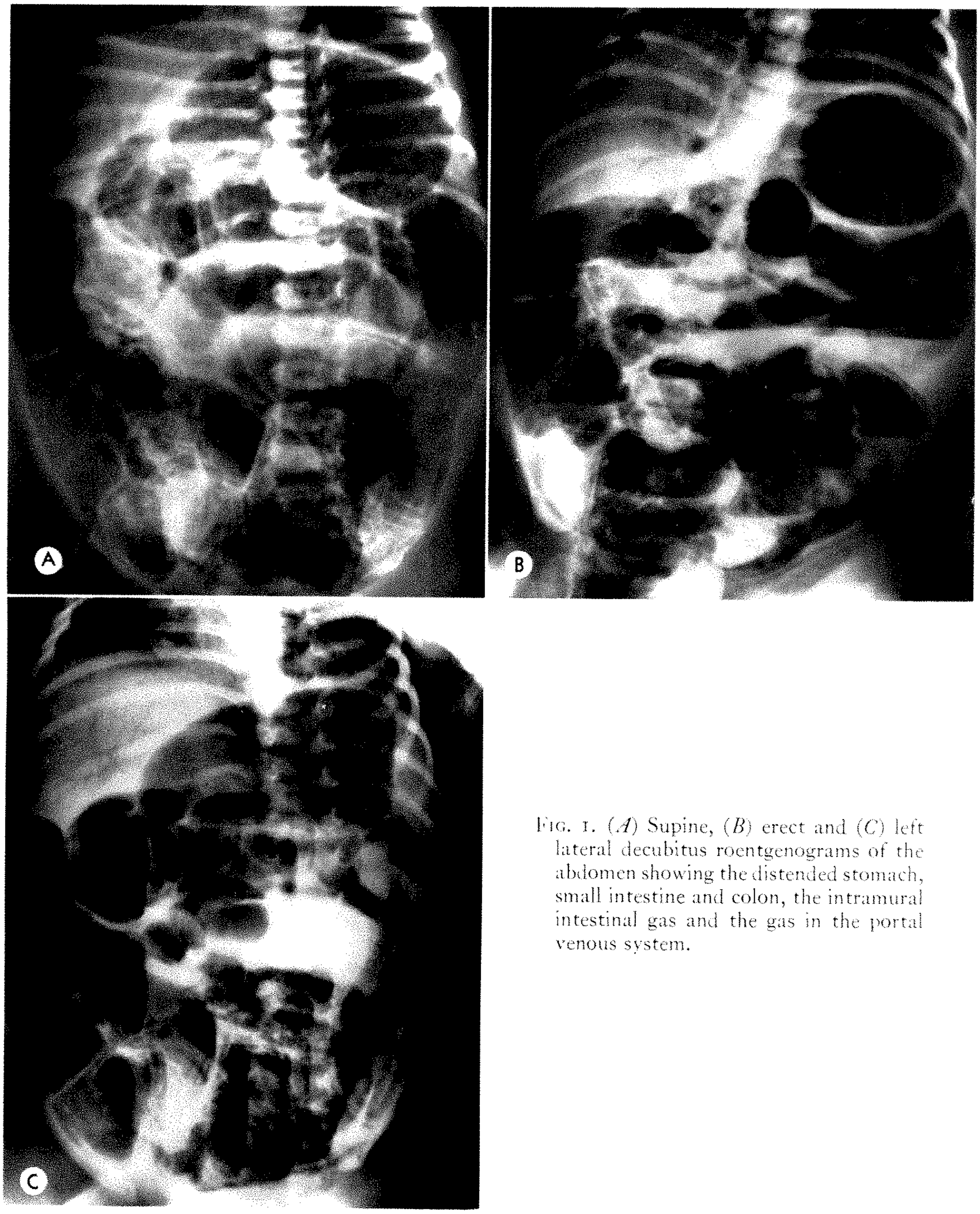

Fig. 1. (*A*) Supine, (*B*) erect and (*C*) left lateral decubitus roentgenograms of the abdomen showing the distended stomach, small intestine and colon, the intramural intestinal gas and the gas in the portal venous system.

vascular occlusion. The many cases of bowel distention without portal vein gas and the cases of portal vein gas with an intact mucosa have been arguments against this proposition. A more popular view suggests that the gas is produced by enteric organisms which have gained access to the vascular system through compromised bowel wall. In both theories, intestinal distention, congestion, and necrosis play an important role.

The roentgen appearance of gas in the

portal venous system is a linear, branching gas pattern in the right upper quadrant radiating outward to the periphery of the liver. This appearance distinguishes it from gas in the biliary tree, with which it is most easily confused and where the gas remains centrally located in the region of the porta hepatis. Once the diagnosis is established, compromised bowel should be suspected. Prompt diagnosis, vigorous antibiotics and supportive therapy, and surgery, if indicated, may result in other survivors.

SUMMARY

A case of portal vein gas in an infant with survival is reported. It is suggested that prompt diagnosis and vigorous therapy may lead to other survivors.

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SIGMOID VOLVULUS IN CHILDREN*

A CASE REPORT

By RICHARD L. LILLARD, M.D.,† R. PARKER ALLEN, M.D.,‡
and J. E. NORDSTROM, M.D.§
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SIGMOID volvulus is encountered so rarely in pediatric practice that its true incidence is not known.[2,8] There have been many reports of this condition in the adult. In the United States, sigmoid volvulus accounts for only 3 to 8 per cent of all intestinal obstructions.[1,5,10] In eastern Europe it is the cause of 30 to 50 per cent of all intestinal obstructions.

The few reported cases in children are probably due to: (1) its rare incidence, about 1 patient in each 4,857 general admissions;[5] (2) low index of suspicion; and (3) the better general status of health, diet and bowel habits of children as compared to adults. Six children with sigmoid volvulus, having an age span of 3 to 11 years, were previously reported from Denver Children's Hospital.[2] All were reduced hydrostatically at fluoroscopy so that surgical proof was lacking.

In this paper, a case is reported of sigmoid volvulus in a 2 year old boy which required surgical reduction when hydrostatic reduction failed.

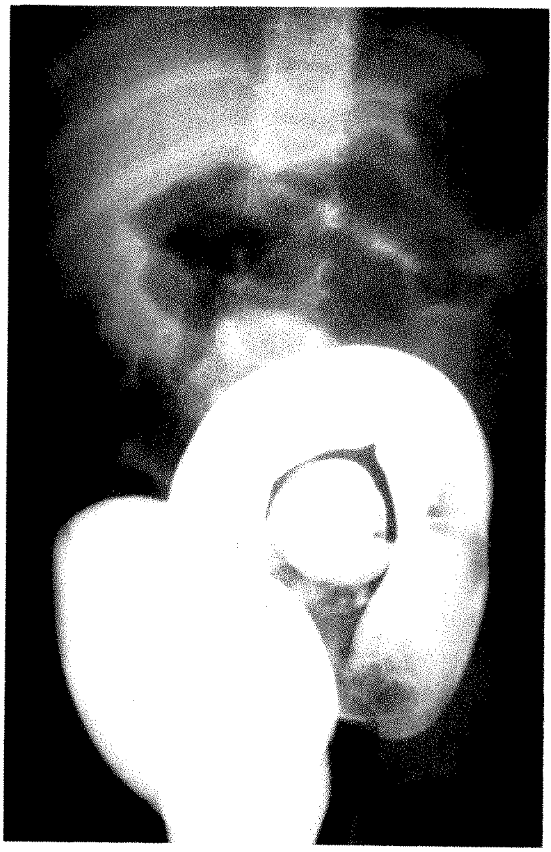

Fig. 1. Introduction of barium demonstrates obstruction at the junction of descending colon with sigmoid. Redundant sigmoid is prominent.

REPORT OF A CASE

This 2 year old male was well until 24 hours preceding admission when he had sudden onset of regular, intermittent abdominal "cramps which seemed like labor pains" according to his mother. He had no emesis or diarrhea, but had concurrent anorexia.

Admission physical findings were not helpful. He had minimal left lower quadrant tenderness. No mass was felt. Distention was equivocal. Rectal examination was negative.

The white blood cell count was 12,800 per ml.; the differential count showed 64 per cent neutrophils, 34 per cent lymphocytes and 2 per cent monocytes. Urinalysis was reported as 2+ acetone, otherwise normal. Bleeding and clotting times were normal.

The tentative clinical diagnosis was intussusception. Barium enema study revealed obstruction at the junction of descending colon with sigmoid (Fig. 1). Following removal of the enema tube and expulsion of some of the barium, air contrast outlined the twisted mucosal pattern and the *tapering deformity of the barium*

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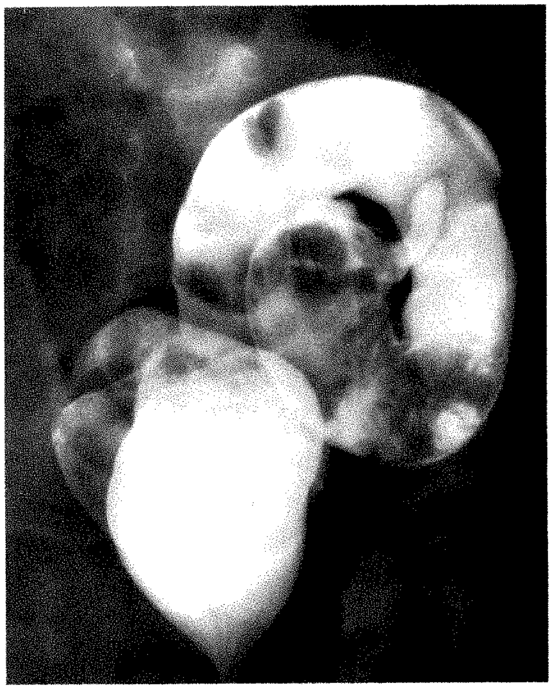

Fig. 2. After removal of the tube, the "twisted taper" deformity in the *proximal* end of the twisted loop is noted.

column which characterizes sigmoid volvulus (Fig. 2). The exact outlines of the markedly redundant colon were evident (Fig. 3).

Two attempts at hydrostatic reduction failed, and surgery was recommended. However, symptoms did abate somewhat and the surgeon requested a plain abdominal roentgenogram which was made 1 hour later. A minute quantity of barium had trickled into the descending and transverse colon (Fig. 4 and 5), but the narrow segment at the proximal end of the sigmoid was still present. Barium was instilled again to determine whether or not spontaneous reduction had occurred. Obstruction to retrograde flow was still complete.

At surgery, a markedly redundant sigmoid colon was twisted clockwise on its mesentery, producing the obstruction.

DISCUSSION

Sigmoid volvulus is most common in the elderly, 70 per cent of patients being over 50 years of age[10] and the peak incidence being at the age of 71 years. Men outnumber women 3 to 1.[7,11]

The predisposing factor common to all patients is a short-based mesosigmoid combined with an abnormally redundant sigmoid colon. This state allows the proximal and distal limbs of the sigmoid colon to lie in close proximity so that the sigmoid colon resembles the Greek omega more nearly than sigma.[5]

The "trigger mechanism" for each case differs.[5] Diet, constipation, mental illnesses and central nervous system diseases have all been implicated.

Whatever the initiating mechanism, torsion of the mesosigmoid occurs, usually in a counterclockwise direction. Simultaneous axial rotation of the *bowel* occurs, equal to twice the mesosigmoid rotation.[6] Sigmoid loop obstruction results (Fig. 4 and 5).

ROENTGEN FINDINGS

Similar findings have been noted in all children with sigmoid volvulus at Denver Children's Hospital. They are as follows:

1. Nondiagnostic bowel pattern on the plain abdominal roentgenogram.
2. Absence of the single sigmoid loop on

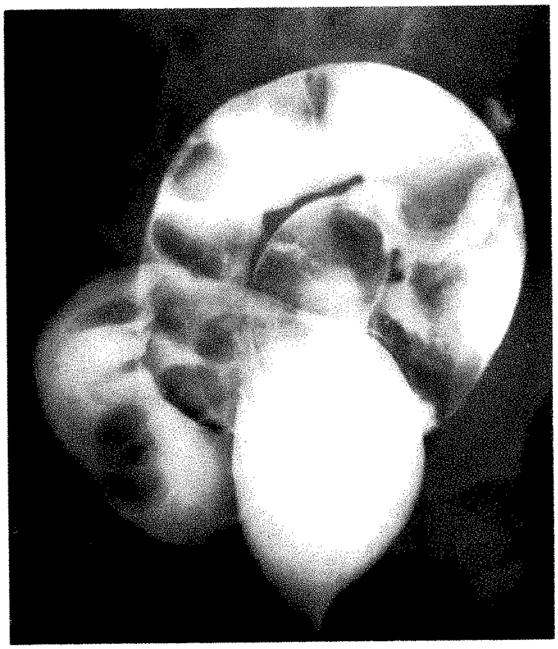

Fig. 3. The redundant loops are crisply outlined by air-contrast study, and a minute quantity of barium has passed the obstructed site.

the plain abdominal roentgenogram which is characteristic in adult patients.

3. Proximal limb sigmoid obstruction as distinct from the distal limb obstruction seen in the adult.

4. Hydrostatic reduction observed fluoroscopically at routine barium enema study.

5. The "twisted taper" configuration of the barium column proximally at the site of mesosigmoid and axial torsion. The term "twisted taper sign" is suggested to best describe the deformity of the barium column at the site of volvulus. This term gives an immediate word-picture, since many candles (tapers) presently available have a design known as "twist tapers." The term is more descriptive of the actual appearance than "bent inner tube," "bird's beak," or "ace of spades," which have been used in the past.

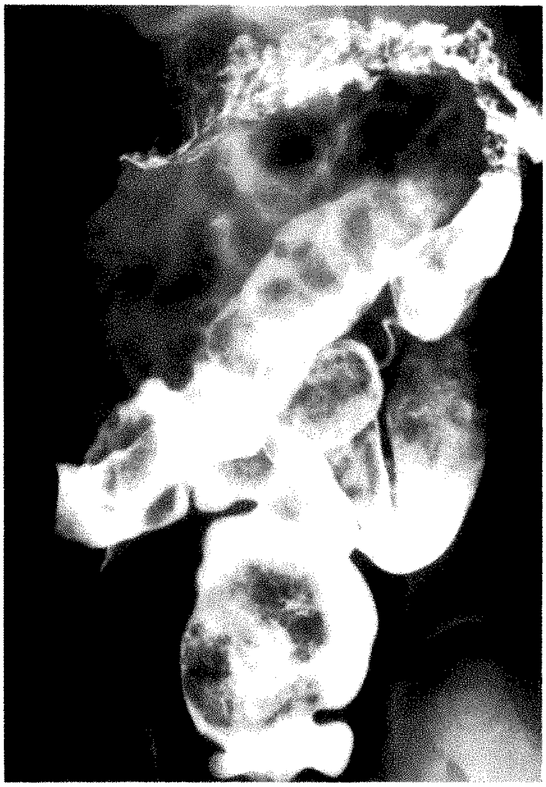

FIG. 4. A repeat examination, approximately 1 hour after the first, demonstrated the narrow lumen at the proximal end of the volvulus. Some barium has leaked proximally.

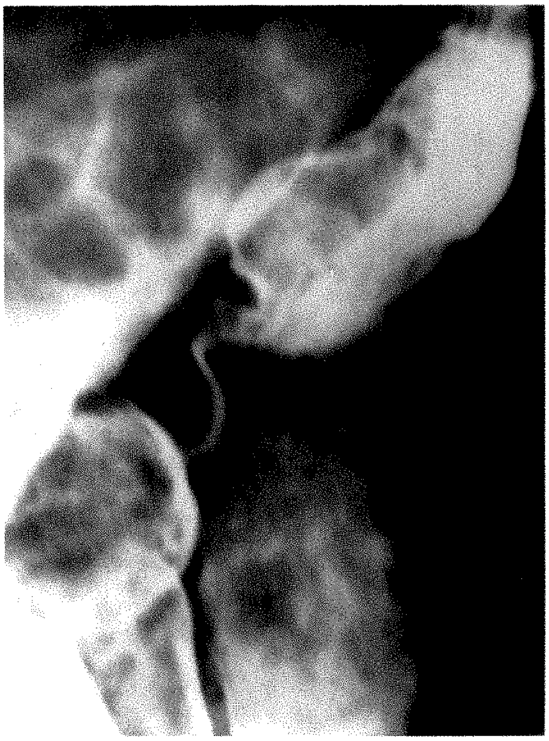

FIG. 5. Close-up of the narrow lumen.

Torsional obstruction at the rectosigmoid junction is characteristic of adult sigmoid volvulus, but obstruction at the junction of descending colon and sigmoid colon is characteristic of the condition in children. Thus, "the twisted taper sign" is produced by distal limb sigmoid obstruction in adults, and by proximal limb sigmoid obstruction in children.

SUMMARY

Sigmoid volvulus is reported in a 2 year old boy in whom an attempt at hydrostatic reduction under fluoroscopic control failed, necessitating surgical reduction. The diagnosis was established preoperatively by roentgen signs, the most important of which was the deformity of the barium column in the proximal limb of the volvulus. The term "twisted taper sign" is suggested as a more descriptive alternative to older terms applied to the mesosigmoid and axial torsion.

Proximal limb sigmoid obstruction is

characteristic of sigmoid volvulus in children, in contrast to the typical distal limb sigmoid obstruction in adults.

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GASTROINTESTINAL FINDINGS IN CYSTIC FIBROSIS*

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CYSTIC fibrosis is a hereditary disease of the exocrine glands. It involves the gastrointestinal tract with differing clinical presentations at ages that range from *in utero* to adulthood. This is, in addition to the chronic pulmonary disease, pancreatic insufficiency and abnormal loss of sweat electrolytes which are characteristic of most patients with cystic fibrosis.

OBSTRUCTIVE MANIFESTATIONS

Approximately 10 per cent of infants born with cystic fibrosis have congenital intestinal obstruction (Fig. 1) due to the inspissation of abnormal meconium (meconium ileus). The abnormal character of the meconium is attributed to abnormal mucus and mucoprotein produced by the intestinal glands and probably, to a lesser degree, to pancreatic achylia.[15] The terminal ileum contains a dried putty-like meconium and is the site of the obstruction. Proximal to this, the ileum is hypertrophied and greatly distended by extremely thick tenacious meconium. The fecal material becomes semifluid still further proximally and the dilatation of the ileum decreases. The roentgen findings in meconium ileus (Fig. 2) where no secondary complications have occurred have been thoroughly reviewed.[10,16] A bubbly appearance may be seen in the right or left side of the abdomen and is not pathognomonic for meconium ileus.

Infants with meconium ileus who survive the initial surgery and hospitalization have the same prognosis as infants with cystic fibrosis beyond the neonatal period (Fig. 3). Meconium ileus in its various forms probably accounts for the major portion of all cases of congenital intestinal obstruction.

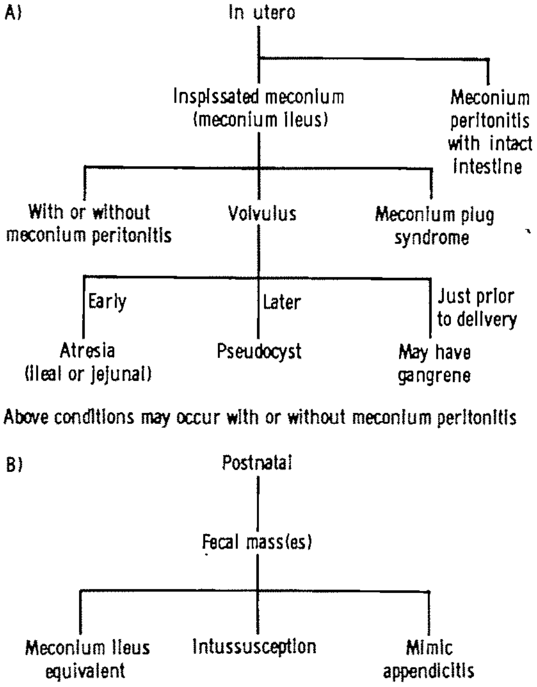
A)
In utero
Inspissated meconium (meconium ileus)
Meconium peritonitis with intact intestine
With or without meconium peritonitis
Volvulus
Meconium plug syndrome
Early
Later
Just prior to delivery
Atresia (ileal or jejunal)
Pseudocyst
May have gangrene
Above conditions may occur with or without meconium peritonitis
B)
Postnatal
Fecal mass(es)
Meconium ileus equivalent
Intussusception
Mimic appendicitis

FIG. 1. Intestinal obstruction in cystic fibrosis.

Volvulus of the ileum occurs in approximately one-half of the infants with meconium ileus. When this occurs just prior to delivery, gangrene may develop if the blood supply to the involved segments of bowel has been compromised for a sufficiently long period of time. If the volvulus happens in the mid to latter part of the third trimester, the loops of ileum become matted together and a gelatinous pseudocyst develops (Fig. 4).

When the volvulus occurs *in utero* early in the last trimester, the matted loops of intestines necrose and become absorbed. Single or multiple atresias develop as a result of the injury to the blood supply (Fig. 5). Air-fluid levels are present early in this form of meconium ileus. The fluid

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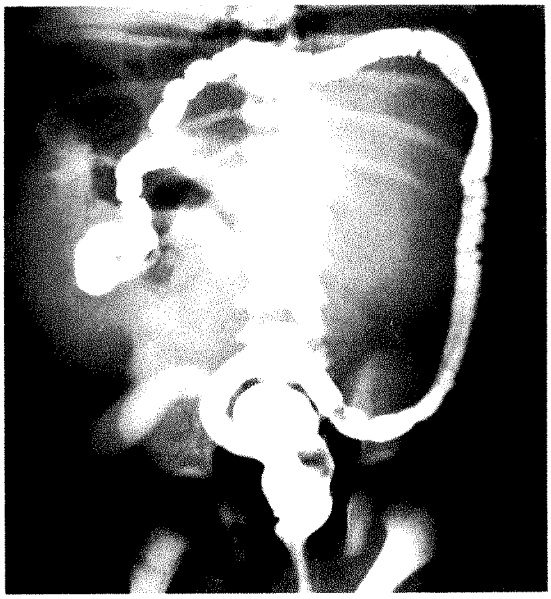

FIG. 2. One day old infant with bile stained vomitus, abdominal distention and obstipation. Barium enema study demonstrates a narrowed caliber colon ("microcolon") and a bubbly pattern in the loops of small bowel in the right upper quadrant. Meconium ileus was found at surgery.

levels are in dilated loops above the blind end of the atresia. The inspissated meconium is usually still present in the remnant of terminal ileum. Intestinal atresia secondary to injury to the fetal intestine has been demonstrated experimentally by Louw and Barnard,[9] and Santulli and Blanc.[14]

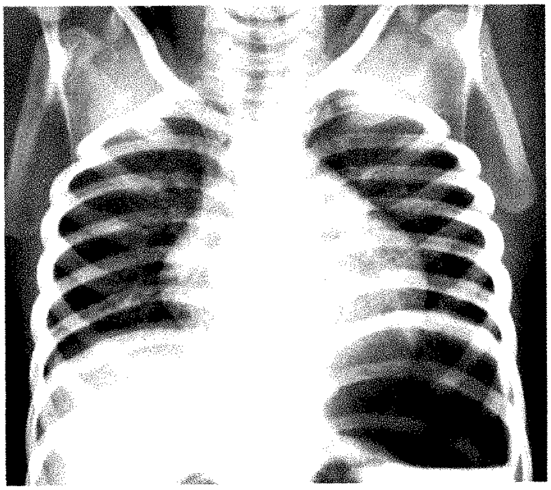

FIG. 3. Same patient as in Figure 2 at 3 years of age. Frontal chest roentgenogram shows the lungs to be clear.

Meconium peritonitis may be present with any of the above complications and results from the *in utero* perforation of the intestine with calcification of the extruded sterile meconium. The calcification may be on the serosal or peritoneal surface or in the wall of the pseudocyst. The perforation is generally sealed over before birth by the local inflammatory process. Meconium peritonitis is present in one-third of the infants with meconium ileus.

The majority of patients with meconium peritonitis are recognized because they have signs and symptoms of congenital intestinal obstruction. The prognosis of these patients is poor. Recently, we examined 3 infants with roentgenographic evidence of meconium peritonitis, but with intact intestinal tracts and no abnormal gastrointestinal symptoms (Fig. 6, *A* and *B*). One patient in addition to peritoneal calcification had lime salts in the scrotum. The intestinal perforation occurred at a

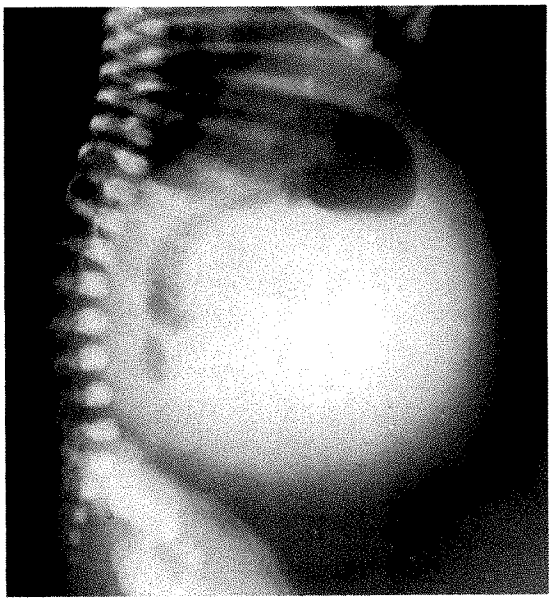

FIG. 4. Newborn with abdominal distention and vomiting. Lateral roentgenogram of the abdomen demonstrates a protuberant abdomen and displaced duodenum and proximal jejunum by a mass. Meconium ileus with a pseudocyst of the ileum was found at surgery.

time when the peritoneal cavity and the processus vaginalis were in communication. All patients were subsequently proven to have cystic fibrosis.

Intestinal glands secreting abnormal mucus and mucoprotein can account for the formation of abnormal meconium, perforation, formation of meconium peritonitis and subsequent sealing over of the perforation site. Since 20 per cent of patients with cystic fibrosis have normal pancreatic enzymes, relatively normal gastrointestinal function can be anticipated as long as obstructive symptoms do not develop as a complication of the perforation.

Some newborn infants have difficulty passing the first meconium stool, with signs of intestinal obstruction. When the meconium is passed, the signs of intestinal obstruction disappear. This has been called the "meconium plug syndrome." This diagnosis should not be made without excluding cystic fibrosis and Hirschsprung's disease, as both of these entities may present as the meconium plug syndrome (Fig. 7).

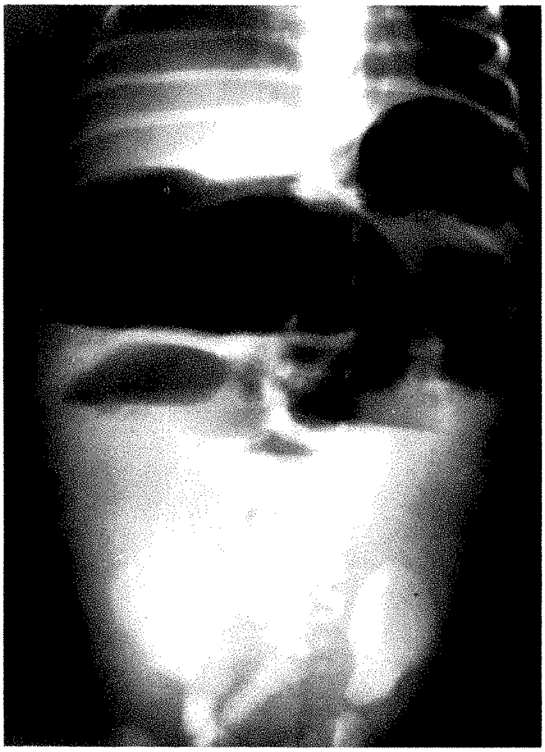

Fig. 5. Newborn with abdominal distention and vomiting. Upright roentgenogram of the abdomen demonstrates air-fluid levels early in the course of meconium ileus. At surgery, inspissated meconium in the terminal ileum and a high ileal atresia were found.

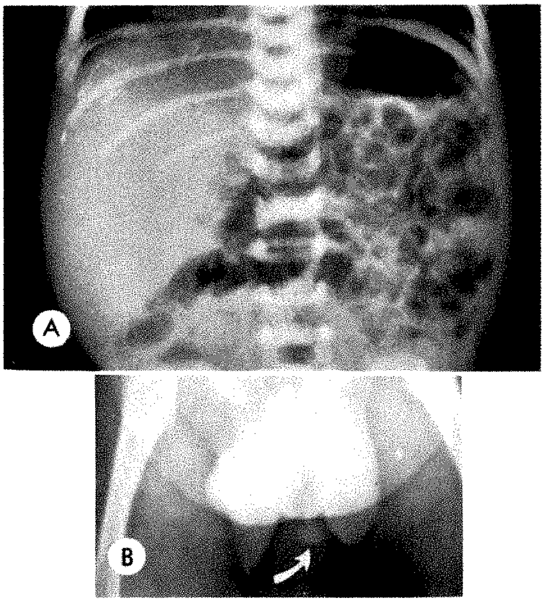

Fig. 6. Roentgenographic studies in a 3 week old infant as part of a work-up to rule out congenital anomalies. A sibling died in infancy of an unknown cause; no autopsy was performed. (*A*) Roentgenogram of the upper abdomen shows calcium under the right dome of the diaphragm. (*B*) Roentgenogram of the scrotum demonstrates a collection of calcium in the scrotal sac. Because of the meconium peritonitis, a sweat test was done. Abnormally high electrolytes were present, confirming the suspected diagnosis of cystic fibrosis.

Inspissated small bowel content causing intestinal obstruction beyond the newborn period has been called "meconium ileus equivalent."[8] Although this complication of cystic fibrosis is infrequently reported,[2] it probably will become more common as survival of these children is prolonged. This complication more often occurs in older children, although we have seen it in a patient only 8 days of age. Instances of this complication have been seen following a change from an uncontrolled to a low fat diet and following inadequate dosage or omission of pancreatic enzymes (Fig. 8; 9; and 10, *A* and *B*).

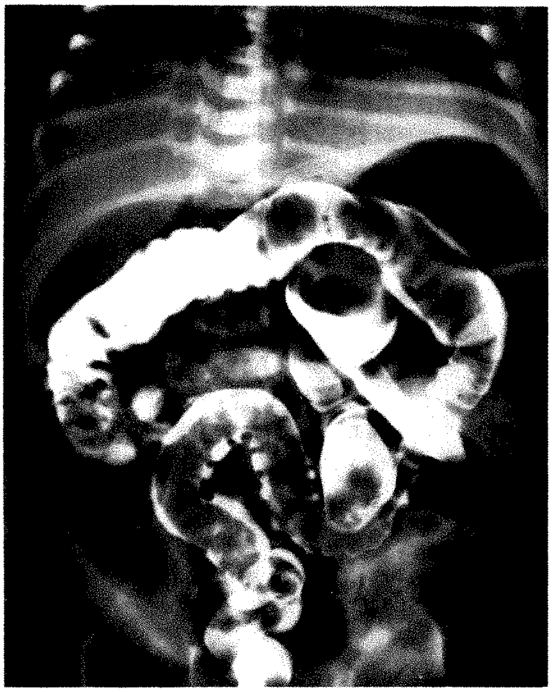

FIG. 7. Thirty hour old infant with progressive abdominal distention since birth. Barium enema study shows a normal caliber colon with a meconium plug from the rectum to the mid-transverse colon. Small bowel distention and intramural air ("pneumatosis intestinalis") are also present. A false negative sweat test was obtained. A meconium cast was passed within 48 hours of age and the abdominal distention subsided. Re-admitted at 2½ months of age for failure to thrive. Diagnosis of cystic fibrosis was established by repeated sweat tests and confirmed at autopsy.

Because of the poor pulmonary status of most of the patients with cystic fibrosis, nonsurgical management would be desirable. When meconium ileus equivalent is present, medical treatment can often be accomplished by administering pancreatic enzymes by both nasogastric tube and enema, and enemas of detergent solutions, saline solutions and mineral oil. Although clinical suspicion of the correct diagnosis should be aroused when mechanical obstruction occurs in a child with cystic fibrosis, the diagnosis is made by roentgenography. Abdominal roentgenograms show a bubbly appearance in the distal small bowel and proximal colon. Contrast enema studies may be necessary to distinguish small from large bowel. A water soluble medium may be preferable in order to avoid inspissation of the barium.

A palpable mass in the right lower quadrant due to inspissated feces is frequently encountered. Severe abdominal pain may be associated, suggesting acute appendicitis. These masses may act as a "leader" for ileocecal or ileo-ileal intussusception. Although the possibility of intussusception has been suspected in many cystic fibrosis cases with abdominal pain, we have demonstrated it infrequently (Fig. 11).

ABDOMINAL DISTENTION WITHOUT INTESTINAL OBSTRUCTION

Marked distention of the abdomen may be noted without intestinal obstruction. Ascites and intestinal gas can present dilemmas in diagnosis (Fig. 12).

Ascites in the newborn is rare. Its association with colon perforation and me-

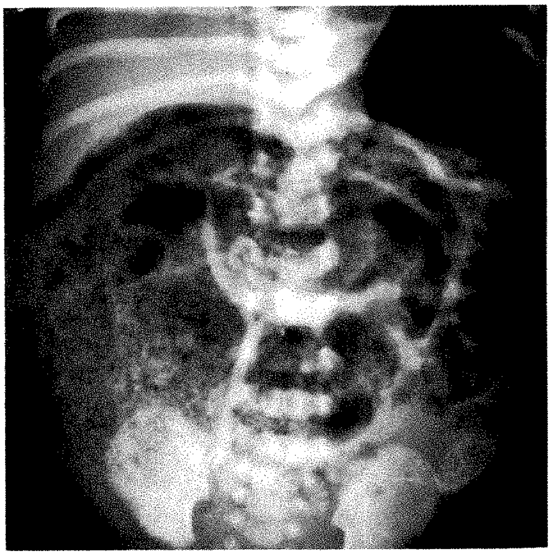

FIG. 8. A newborn with a family history of cystic fibrosis had surgery for intestinal obstruction due to meconium ileus. The meconium was removed and an ileostomy was performed. One week later the abdomen again became distended and a roentgenogram showed dilated small intestine with the bubbly fecal content in the small intestine of "meconium ileus equivalent." The ileostomy stoma is on the left lower quadrant of the abdomen.

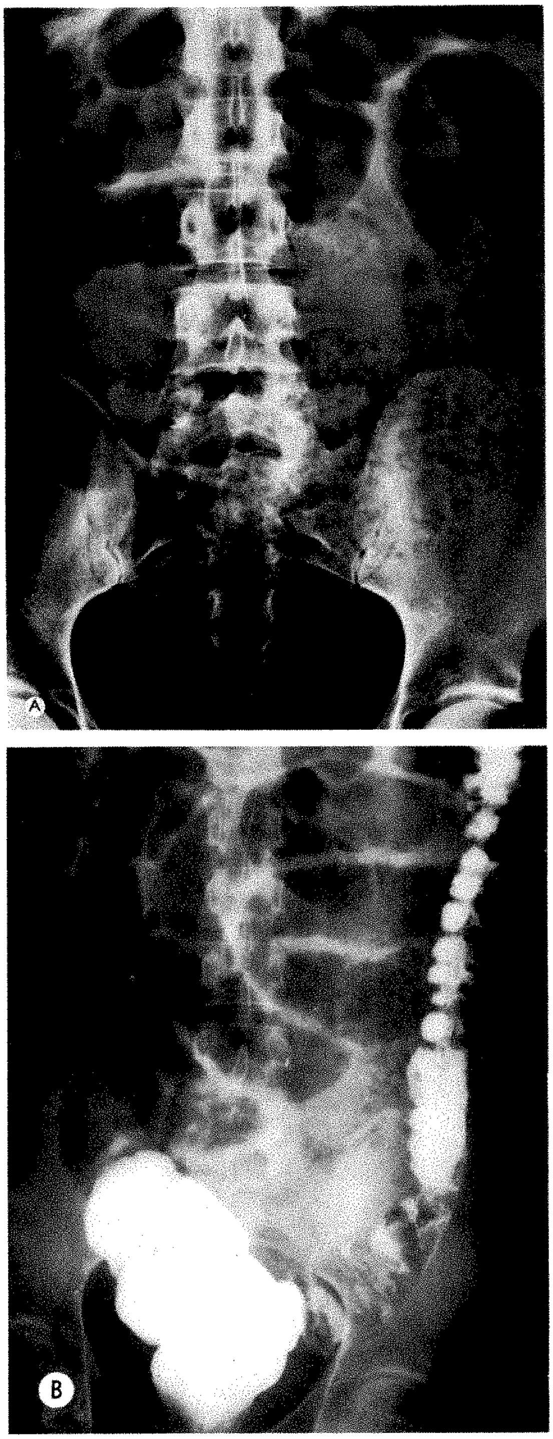

FIG. 10. An adult male in his twenties with known cystic fibrosis since childhood had eliminated pancreatin from his diet for several years. He was admitted for crampy abdominal pain, vomiting, and constipation. (*A*) Abdominal study demonstrates a bubbly pattern in the left lower quadrant. (*B*) Barium enema study shows that the bubbly pattern is in the small intestine. Many loops of dilated small intestine are seen. The obstruction was relieved by detergent enemas.

conium peritonitis is even more uncommon. The ascites in the patient shown in Figure 13, *A* and *B* is presumably from swallowed amniotic fluid, in spite of the perforation being so distal in the intestinal tract. The perforation site on the surgical specimen was interpreted as an anoxic ulcer. Meconium ileus or a volvulus was not present. The colon perforation was probably a very late *in utero* complication and not directly

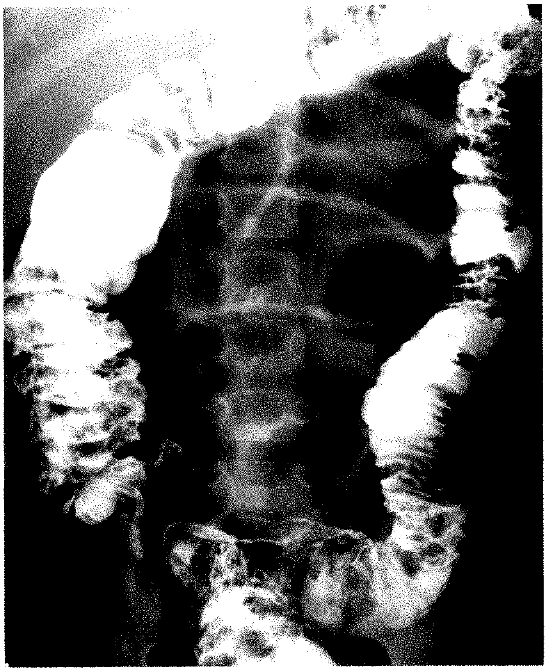

FIG. 9. A 9 year old male with known cystic fibrosis sporadically omitted pancreatic enzymes from his meals. Obstipation developed, associated with abdominal pain. Barium enema study demonstrates feces throughout the colon and a bubbly pattern in the distal small bowel. Pancreatic enzyme and detergent enemas relieved the obstruction and the dilatation of the small intestine subsided.

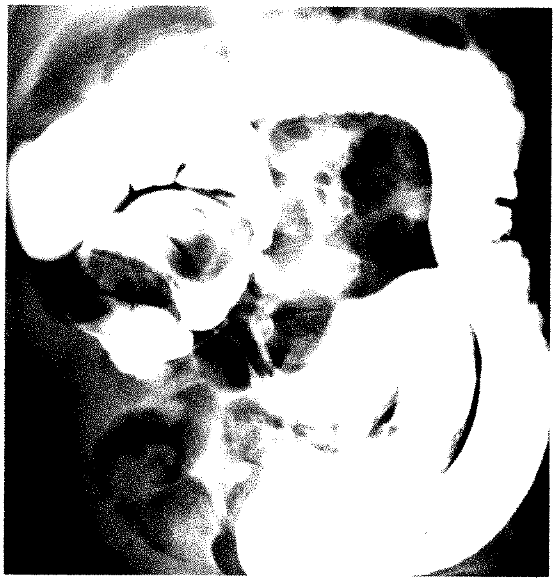

FIG. 11. A 14 month old infant on no pancreatic enzyme supplementation. Intermittent constipation since birth. Barium enema study showed an ileocecal intussusception due to a fecal mass in the terminal ileum acting as a "leader." The intussusception was reduced and fecal masses were passed with detergent enemas.

related to the meconium peritonitis, which probably was an earlier event.

Hypoproteinemia due to malabsorption or cirrhosis of the liver may cause ascites. Infants unsuspected of having cystic fibrosis often have their formulas changed when diarrhea develops. Soy bean formula which is less allergenic than cow's milk is often used. This source of protein has been found to be poorly utilized by infants with cystic fibrosis and has caused hypoproteinemia and anasarca (Fig. 14, *A* and *B*) in infants subsequently proven to have cystic fibrosis. Human milk has a low protein content compared to cow's milk and it too may be more apt to lead to hypoproteinemia.[6] These infants are frequently admitted to the hospital for evaluation of congenital nephrosis which can be ruled out by a normal urinalysis and serum cholesterol. Although fewer than 30 cases have been reported, 10 patients with this complication have been seen at Babies Hospital. None of these patients was found to have a protein losing enteropathy. Cystic fibrosis should be considered in any infant with edema and a normal urinalysis. However, with edema, false negative sweat tests may be obtained.

Ascites secondary to cirrhosis and hypoproteinemia may be the presenting symptom in a child with cystic fibrosis (Fig. 15). Focal biliary cirrhosis is commonly seen at autopsy.[3] The majority of these patients die of respiratory complications and do not have clinical or laboratory findings to suggest liver abnormalities. As multiple foci merge, a multilobular biliary cirrhosis with portal hypertension develops. This complication occurs in approximately 2 per cent of patients with cystic fibrosis (Fig. 16, *A* and *B*). As the liver attempts to regenerate, an enlarged nodular liver forms which may cause pressure irregularities along the duodenal sweep (Fig. 17).

The biliary cirrhosis is initiated by inspissation of bile ductules, presumably by abnormal secretory products of exocrine glands. Cystic fibrosis is not an all or none disease and the involvement of the liver provides another example of the variability of this disease. Several of the patients with severe cirrhosis have had very mild pulmonary disease. Icterus has been absent. Ascites associated with cirrhosis of the liver, especially in an older child, should suggest the possibility of cystic fibrosis.

Diarrhea in infancy is often associated with considerable gas in the intestinal tract and abdominal distention. Total colon aganglionosis generally presents the roentgenographic findings of a normal caliber, relatively shortened colon. The neonatal history in these patients is one of bile

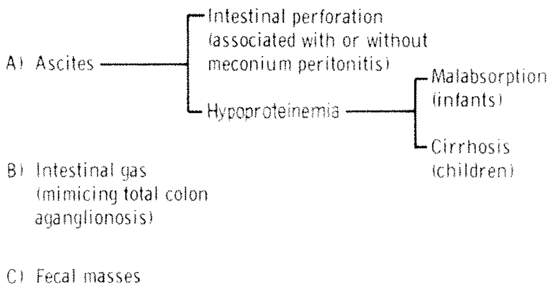
A) Ascites
Intestinal perforation (associated with or without meconium peritonitis)
Hypoproteinemia
Malabsorption (infants)
Cirrhosis (children)
B) Intestinal gas (mimicing total colon aganglionosis)
C) Fecal masses

FIG. 12. Abdominal distention without intestinal obstruction.

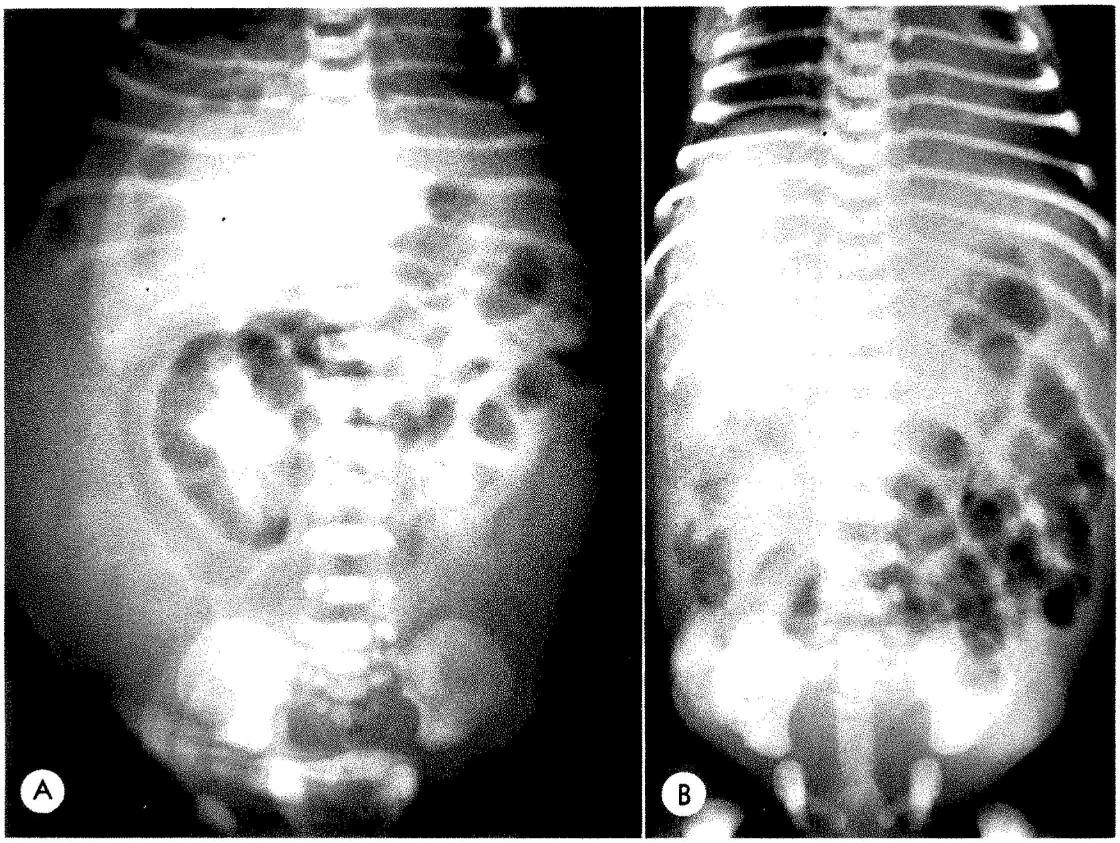

FIG. 13. A newborn with marked abdominal distention at birth. Ascites was noted on plain roentgenograms. (*A*) An intravenous pyelogram shows the liver to be well outlined due to the "bodygram" effect. Small intestinal loops are "floating" in the ascitic fluid. Air bubbles are in the right upper quadrant. At surgery, a perforation of the transverse colon was found and was considered to be due to an anoxic ulcer. (*B*) The postoperative roentgenogram of the abdomen clearly shows calcium in the peritoneum (meconium peritonitis) which was not well visualized preoperatively.

stained vomitus.[1] Intermittent diarrhea is common. An infant with the findings on barium enema examination suggesting total colon aganglionosis (Fig. 18, *A* and *B*), but not vomiting bile during the first week of life, stresses the importance of the neonatal history.

The pathogenesis of other lesions in the intestinal tract of patients with cystic fibrosis is less well understood than that already described. Gastrointestinal examination, using nonflocculating barium, usually demonstrates a normal mucosal pattern and caliber. However, we have observed several older children with a bizarre pattern (Fig. 19, *A* and *B*) of markedly thickened and distorted mucosal folds, and mildly dilated loops which were not edematous or separated. Three of the children demonstrated severe involvement from the ligament of Treitz to the rectum. Five additional children were noted to have less marked mucosal changes and localized disease in segments of small or large intestine. A "cobblestone" appearance of the mucosa of the colon has been described,[7,16] but involvement in the small bowel has not been previously reported.

The pattern may resemble that seen in patients with lymphangiectasia of the intestine (protein losing enteropathy), but edema of the bowel wall and separated loops are present in this disease, differentiating it from cystic fibrosis. Acanthocytosis (β-lipoprotein abnormality) may mimic this pattern, but is easily differentiated

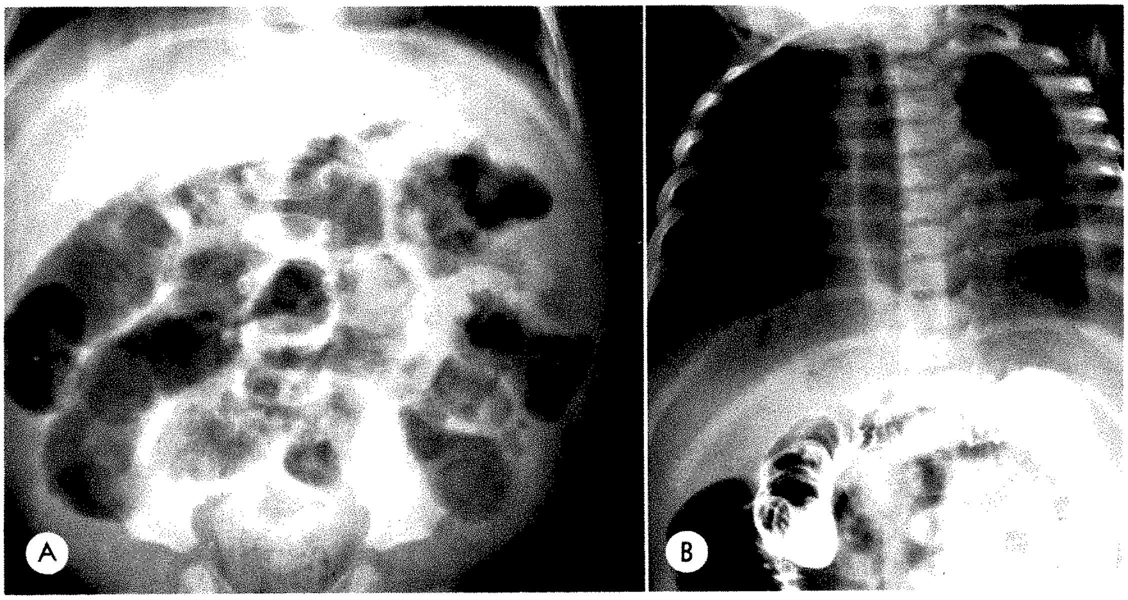

Fig. 14. A 10 week old infant admitted with anasarca and a presumptive diagnosis of congenital nephrosis. The infant was on a soy bean formula for diarrhea. (*A*) On abdominal roentgenogram the flanks are protuberant and the loops are separated by bowel wall edema and ascites. (*B*) Lateral decubitus roentgenogram shows a moderate accumulation of pleural fluid. In view of normal urinalyses, cystic fibrosis was considered as a possible cause of the anasarca and this was proven by abnormal sweat electrolytes.

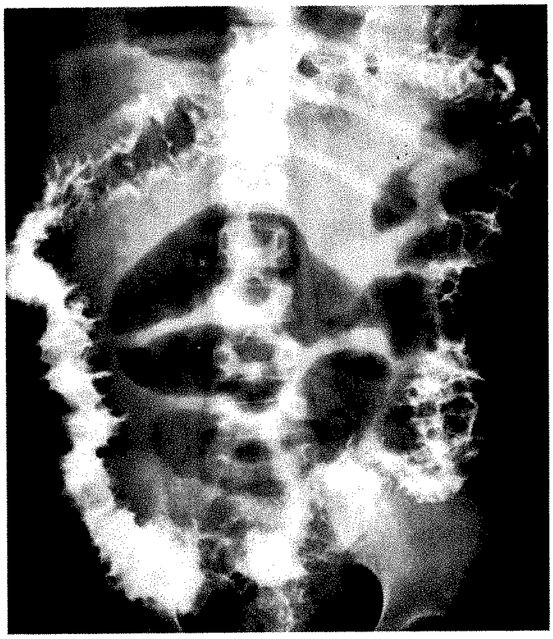

Fig. 15. A 9 year old female was seen for the gradual enlargement of her abdomen; she was otherwise asymptomatic. Physical examination revealed ascites and a large, nodular liver. On the post-evacuation barium enema roentgenogram, the mucosa of the colon is edematous and loops of small bowel are separated. Diagnosis of cystic fibrosis was established by abnormal sweat electrolytes.

from cystic fibrosis by the characteristic spurring of the red blood cells seen on a blood smear.[13]

Thus far, no correlation has been found between this abnormal pattern in the intestinal tract and cirrhosis of the liver or hypoproteinemia. Abnormal mucoproteins of the duodenal juices[5] and malabsorption of both neutral fats and fatty acids[11] indicate abnormal secretions of intestinal glands in addition to pancreatic dysfunction. Histologically, variations from the normal are found in the intestinal tract.[15] What role these abnormalities may play in the pathogenesis of this bowel pattern is not known since adequate biopsies have not been done on our patients to date.

Diabetes has been encountered in pa-

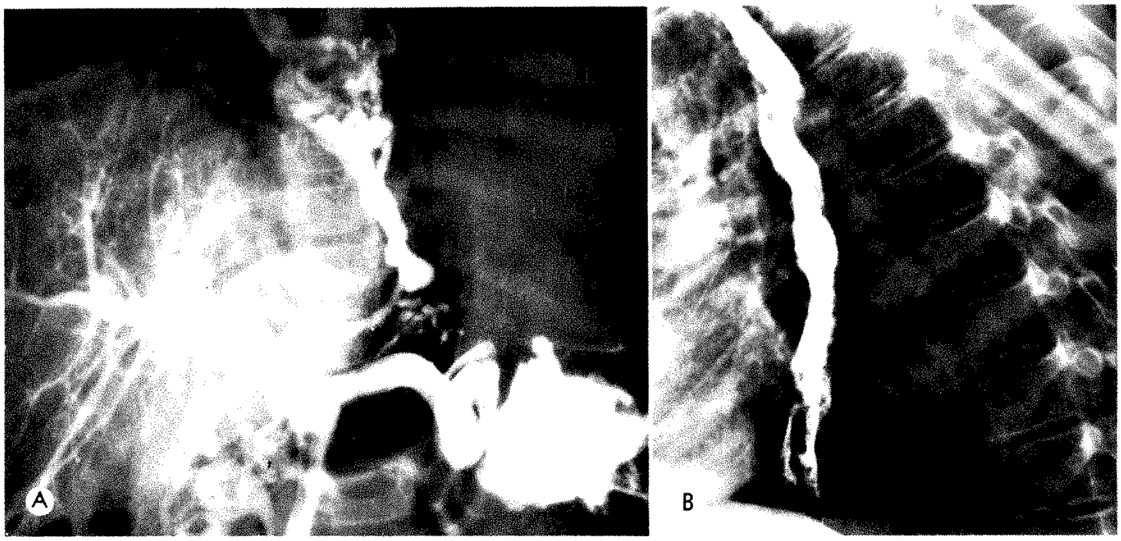

Fig. 16. (*A*) Splenoportogram of a child with cystic fibrosis demonstrating esophageal varices. (*B*) Lateral chest roentgenogram with barium in the esophagus shows filling defects due to the tortuous esophageal veins. Chronic pulmonary disease is also present.

tients with cystic fibrosis. Di Sant'Angnese and Lepore[4] reported 2 children from Babies Hospital with diabetes in over 600 children with cystic fibrosis. These patients were 10 and 13 years of age at the onset of diabetes. One child had a family history of diabetes. Abdominal roentgenograms demonstrated multiple flecks of calcifications in the pancreas (Fig. 20) of both patients, a most unusual finding in cystic fibrosis. Rosan *et al.*[12] discovered 10 of 1,000 children with cystic fibrosis to have diabetes, only 1 had roentgenographic evidence of calcification in the pancreas.

Many patients with cystic fibrosis have died in early infancy, before diabetes would be expected to develop. It is likely that diabetes with or without pancreatic calcifications may be encountered more frequently as the average age of the patients increases. A higher incidence of diabetes has been noted in relatives of patients with cystic fibrosis; the reason for this is unexplained.

Duodenal ulcers are found more frequently at autopsy than in clinical experience. On theoretic grounds, an association of duodenal ulcers and cystic fibrosis would be expected because of the lack of buffering of gastric acid by duodenal pancreatic secretion. Acidosis due to chronic respiratory insufficiency, especially in the terminal months, may play a role in the pathogenesis of the ulcers found at autopsy. The number of cases of duodenal ulcers demonstrated ante mortem in children with cystic fibrosis is small (Fig. 21).

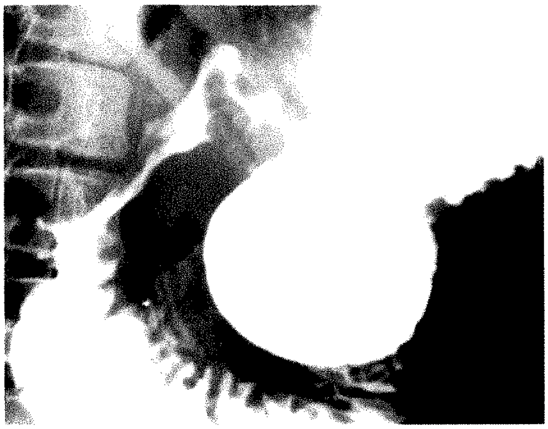

Fig. 17. A 12 year old female with known cystic fibrosis since infancy. Her pulmonary disease has been under good control, but cirrhosis of the liver has been noted for 2 years. Upper gastrointestinal study shows that the first portion of the duodenum is indented irregularly by the enlarged, nodular liver.

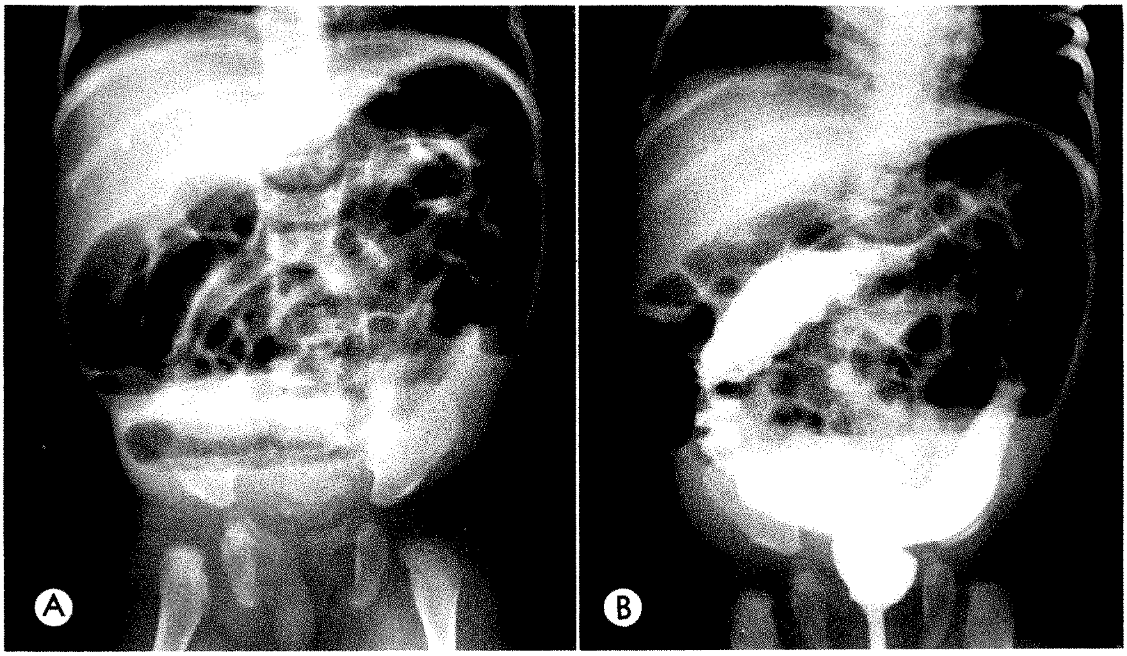

Fig. 18. A 3 month old infant with intermittent diarrhea since birth and an erroneous history of bile stained vomitus, which led to the suspicion of total colon aganglionosis. (*A*) On the upright roentgenogram of the abdomen, dilated small intestine with air-fluid levels is present. (*B*) Barium enema study shows a normal caliber, slightly shortened colon. Rectal biopsy was normal, excluding aganglionosis. Because of the diarrhea, a sweat test was done and the diagnosis of cystic fibrosis was established.

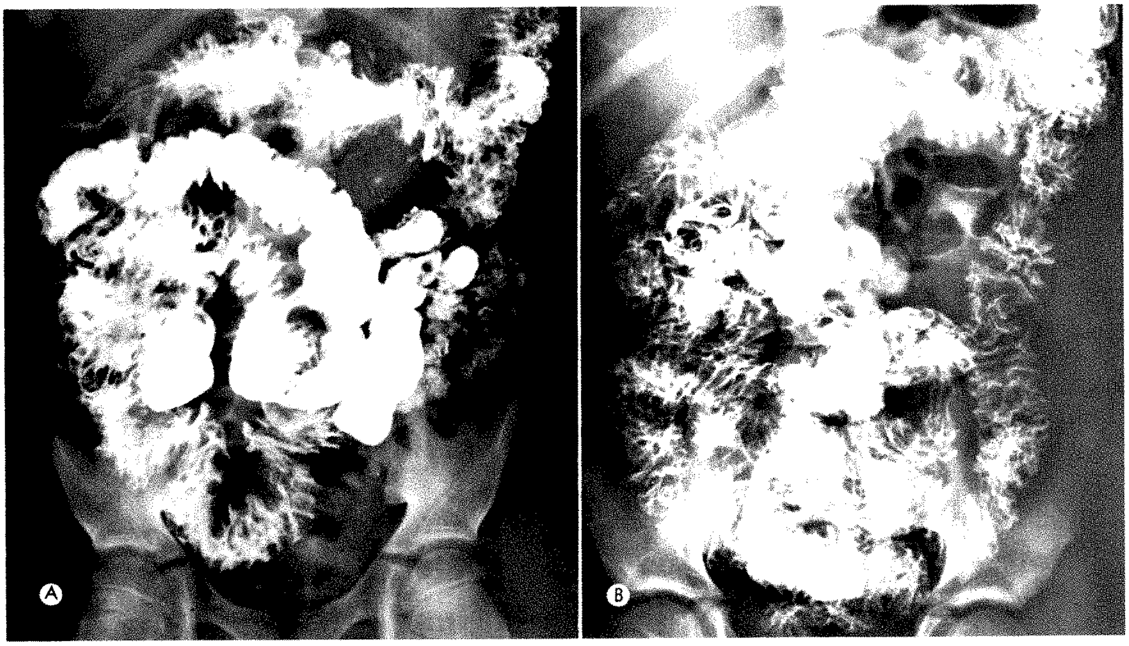

Fig. 19. An 8 year old male with cystic fibrosis diagnosed in early childhood had intermittent, generalized abdominal pain. (*A*) Small bowel study demonstrates an erratic mucosal pattern with mild dilatation of the loops of intestine. (*B*) The postevacuation barium enema roentgenogram shows an irregular mucosal pattern similar to that in the small intestine.

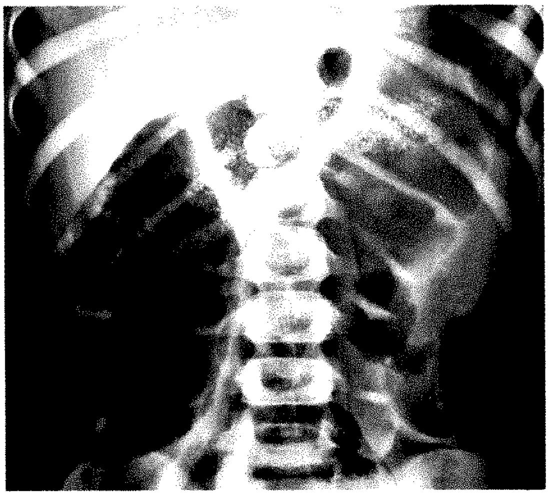

Fig. 20. A 10 year old female with known cystic fibrosis since early childhood. Abdominal roentgenogram shows multiple calcifications in the pancreas at the time the diabetes was recognized.

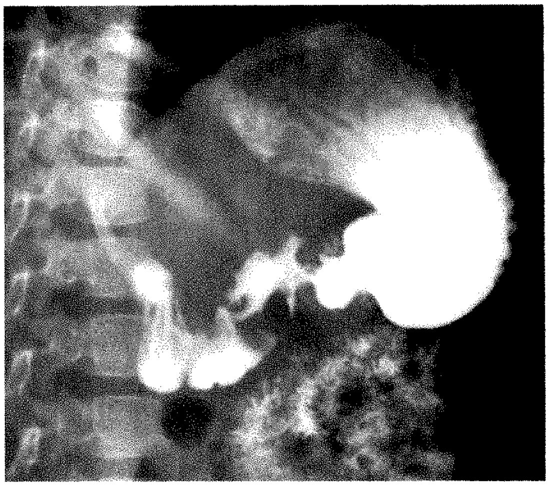

Fig. 21. A chronically ill 8 year old male with epigastric pain for several months and one episode of hematemesis. Upper gastrointestinal study demonstrates a large postbulbar ulcer.

SUMMARY

1. Cystic fibrosis is a generalized disease due to dysfunction of exocrine glands. Gastrointestinal manifestations may occur from *in utero* to adulthood.

2. Meconium ileus and secondary complications such as pseudocysts and atresias are the major causes of congenital intestinal obstruction.

3. Edema may be the presenting symptom due to hypoproteinemia. This may be secondary to malabsorption in the infant and due to cirrhosis in the older child.

4. Inspissated small bowel contents after the newborn period may act as a "leader" in intussusception, simulate acute appendicitis, or cause intestinal obstruction ("meconium ileus equivalent").

5. The pathogenesis of a bizarre pattern in the small and large intestine, calcification of the pancreas and duodenal ulcers are less well understood than the other gastrointestinal findings presented.

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ANGIOGRAPHIC EVALUATION OF ANURIA IN THE NEWBORN[*]

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ANURIA in the newborn is common during the first 24 hours of life, being present in about 30 per cent of the infants.[8] When persistent for a longer period of time, it is usually due to such severe renal dysplasias as renal agenesis and polycystic disease or complete obstruction of the urinary tract. It is important to diagnose definitively the presence or absence of the occasional surgically correctable lesion. Suprapubic puncture of the bladder, when positive, is an aid in making a diagnosis. Retrograde femoral aortography, however, is a relatively simple and precise method of determining the presence of renal parenchyma. Recently, such a procedure performed on an anuric infant quickly established a definite diagnosis of bilateral renal agenesis.

REPORT OF A CASE

I.H. (No. 33-83-90), a 3 day old, 1,785 gm. premature female of about 30 weeks' gestation, was transferred to University of Oregon Medical School Hospital for evaluation. The infant became tachypneic 2 hours after delivery, but was not cyanotic; a chest roentgenogram was negative. Failure to void was soon noted; the blood urea nitrogen was 65 mg. per cent. The suprapubic tap failed to recover urine. On admission, the infant's respirations were labored. The ears were deformed and low-set; there was edema about the eyes, and the left hip was dislocated. A urethral dimple was present, but the urethra was not patent. The provisional differential diagnosis was bilateral ureteropelvic obstruction, urethral atresia or congenital renal agenesis. Emergency abdominal aortography was performed (Fig. 1, *A* and *B*), and a diagnosis of congenital bilateral renal agenesis was established. The infant expired of respiratory failure at 6 days of age. The blood urea nitrogen was 98 mg. per cent. Autopsy revealed complete absence of kidneys, renal arteries, ureters, urinary bladder and urethra.

COMMENT

The reported incidence of bilateral renal agenesis is about 1:4,000 births; 70 per cent are males.[4] Most often these infants are premature. If carried to term, little weight is gained after the 32nd week. The majority dies of respiratory failure within a few hours. One infant has reportedly lived for 39 days.[7]

EMBRYOLOGY

A normal kidney is formed only when a ureteral bud originates from the mesonephric duct and penetrates metanephrogenic mesoderm. The stimulus of the branching ureteral bud is essential for the differentiation of this nephrogenic cap[5] (Fig. 2). Apart from rare hereditary abnormalities, the primary cause of dysplasia is developmental inhibition or arrest.[1] The fetal kidney forms between the 5th and 7th weeks. It is during this period that the developing kidney is vulnerable to environmental influences such as nutritional deficiencies and endocrine disturbances, or to infectious, chemical (drugs) and physical agents. Abortogenic drugs such as the protoplasmic poison quinine may be a significant cause.

If the ureteric buds do not form, the metanephros (kidney) will not develop. The absence of fetal urine results in oligohydramnios, which in turn leads to positional abnormalities of the extremities and pulmonary hypoplasia. A characteristic facial appearance is said to accompany the absence of renal function. "The face is characterized by a prominent epicanthic

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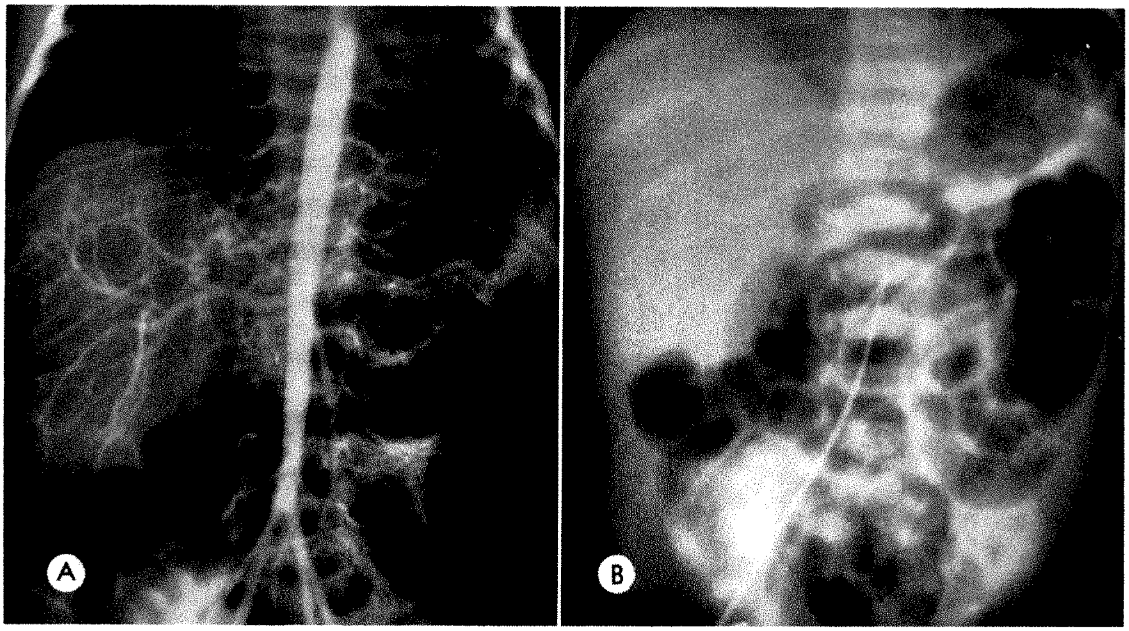

Fig. 1. Abdominal aortograms. Serial roentgenograms were obtained following a hand injection of 3 cc. of 60 per cent conray through a PE 120 catheter inserted via the right superficial femoral artery. (*A*) Arterial phase. All of the usually recognized branches of the abdominal aorta except the renal arteries can be identified. (*B*) Nephrographic phase. A nephrogram is not observed.

fold, a flattened nose, large, imperfectly formed ears set low on the head and often less upright than normal, and an unpleasant, senile expression" (Potter's syndrome).[4]

At least 85 per cent of infants with bilateral renal agenesis have typical facial characteristics.[2] This is related to the absence of kidney function rather than to a specific malformation. Any infant with a severe urinary tract dysplasia or anomaly, therefore, may present a similar appearance.

DISCUSSION

The differential diagnosis of an anuric

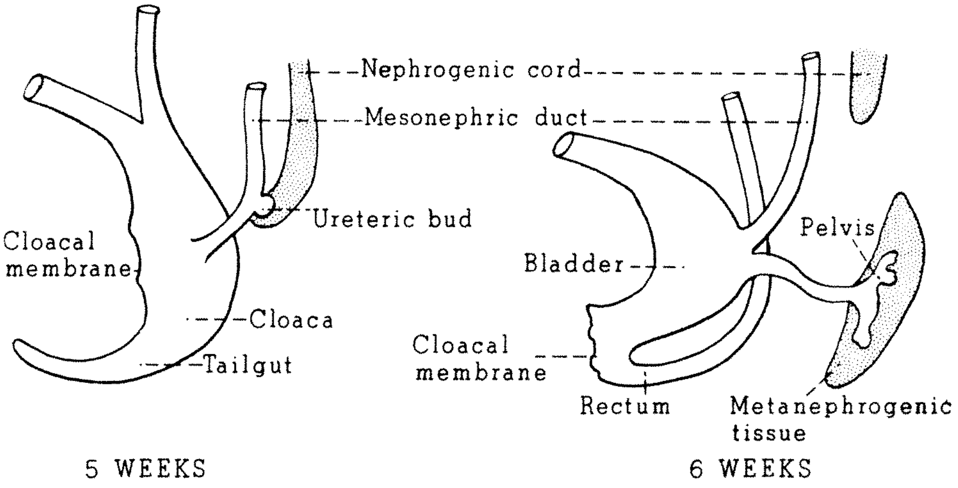

Fig. 2. Diagram showing the early stages of the embryonic development of the urinary tract (after Arey[1]).

state in a newborn includes such severe dysplasias and anomalies as renal agenesis, polycystic disease, urethral atresia, and ureterovesical or ureteropelvic obstruction. The case reported did not present entirely typical Potter's facies. Because the urethra was not patent, it was possible that an obstructive uropathy was present. When there is a possibility that a surgically correctable lesion is present, aortography can be used to determine quickly the presence of renal arteries or parenchyma. Even if the diagnosis is bilateral renal agenesis, the clinician will be able to advise the parents of the prognosis and can follow a conservative treatment plan.

SUMMARY

The differential diagnosis of anuria in the newborn over 24 hours of age includes severe renal dysplasia and complete urinary obstruction. The latter is rare, but may be correctable. When it is reasonable to expect that significant information can be gained, aortography is indicated and feasible even in a premature infant.

The use of angiography to establish the diagnosis of bilateral renal agenesis in a 1,785 gm. infant is reported.

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CONGENITAL INDIFFERENCE TO PAIN*

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THE condition of congenital indifference to pain was first reported by Dearborn[4] in 1932 as a case of "congenital general pure analgesia." The term was applied to a 44 year old man who could tolerate penetration of a large number of needles and who exploited this disorder by advertising himself as a "human pincushion." Subsequent recognition of a small number of individuals with congenital indifference to pain has resulted in excellent articles on this subject.[3,8,11] The condition is relatively rare; to our knowledge less than 50 authenticated cases have been recorded.

A patient exhibiting the typical manifestations of the disorder has been followed at the Montefiore Hospital and Medical Center for the last 8 years. This report is a brief exposition of the salient features of the entity with emphasis upon the characteristic roentgenologic manifestations.

REPORT OF A CASE

R.V. was a product of a normal full-term pregnancy and a spontaneous delivery. In the first year of life (1950), he was admitted to the Neurological Institute of the Columbia Presbyterian Medical Center in New York City, with a history of bleeding from intraoral ulcerations. An area of swelling was present in the right parieto-occipital area. Several fingers showed scars due to burns. A neurologic consultant mentioned the fact that the child felt no pain. There were no other neurologic abnormalities. A fissure fracture of the right occipital bone was present. A combined ventriculogram and pneumoencephalogram revealed no abnormality.

In 1953 the patient was described by his mother as a rough and aggressive child who had continued to sustain burns and bruises without complaint. The child earned a rating of "bright, normal intelligence" on psychometric testing. No abnormality was noted in a careful evaluation of cranial nerves, coordination and motor function. Temperature, light touch, and position senses were intact. There was no reaction to pressure on the Achilles tendon or testicles.

At the age of 6 years he was seen at Montefiore Hospital with fever and a swollen left knee. Physical examination revealed a scarred left knee. The joint was swollen and tense and there was limitation of flexion. Aspiration yielded 50 cc. of cloudy yellow fluid. A roentgenographic examination of the knees was obtained (Fig. 1, *A* and *B*; and 2, *A* and *B*).

Although osteomyelitis was suspected, a bone tumor could not be excluded with certainty and a biopsy of the left medial femoral condyle was performed. The specimen revealed chronic inflammatory granulation tissue.

A follow-up examination of the right knee was obtained at the age of 9 years (Fig. 3, *A* and *B*). The patient was last seen in the Orthopedic Department of Montefiore Hospital at the age of 14 years. Physical examination showed a well developed white male, height 61.5 inches, weight 106 pounds. There was marked scarring of the tongue and advanced carious tooth decay. Both knees and ankles were deformed. The right knee was ballotable and small bone fragments were palpable on the lateral inferior surface of the patella (Fig. 4, *A* and *B*). An arthrotomy of the right knee was performed. The synovium was markedly thickened. Multiple loose bodies were removed and a lateral meniscectomy and synovectomy were accomplished. Roentgenographic study of the right ankle was also obtained. This revealed neuropathic changes involving the talonavicular joint (Fig. 5). The patient recovered uneventfully from knee surgery and is attending high school.

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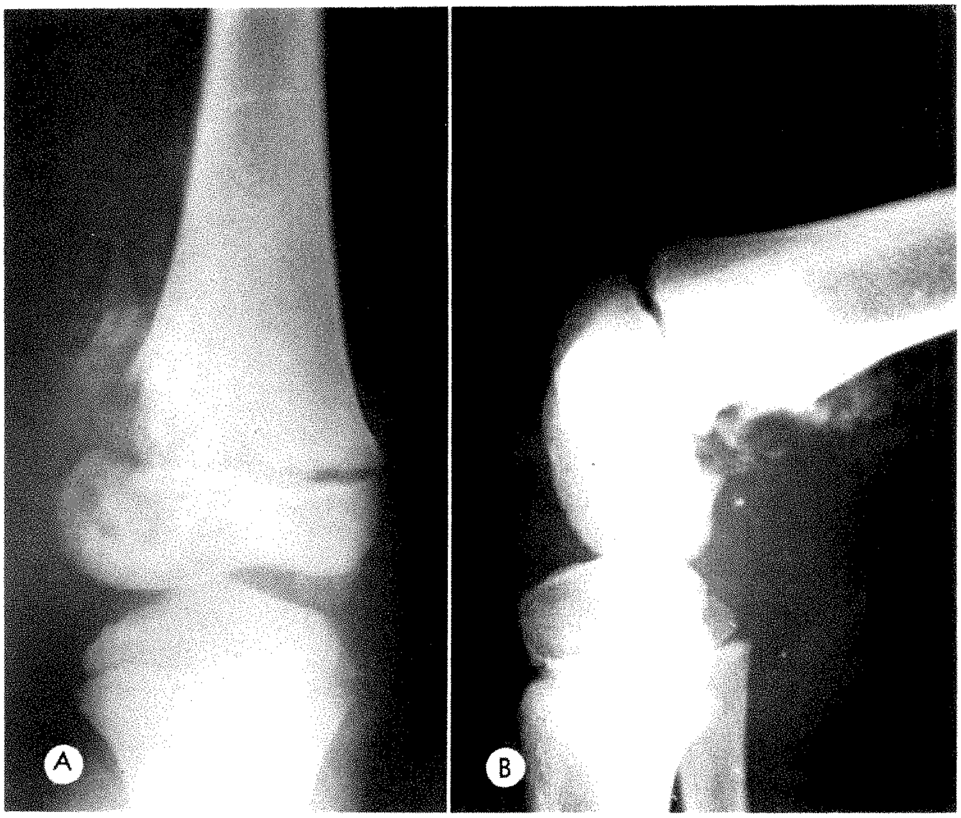

Fig. 1. *Left knee at age of 6 years.* (*A* and *B*) Femoral metaphyseal focus of osteomyelitis.

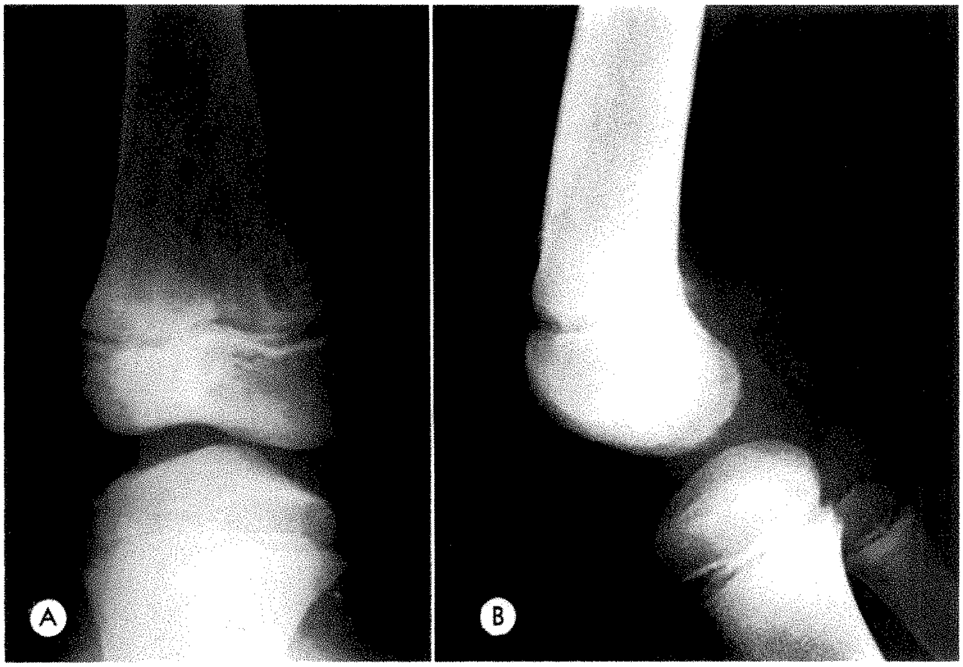

Fig. 2. *Right knee at age of 6 years.* (*A*) Anteroposterior study showing lucent femoral metaphyseal inflammatory focus. (*B*) Lateral study showing early aseptic necrosis of the lateral femoral condyle.

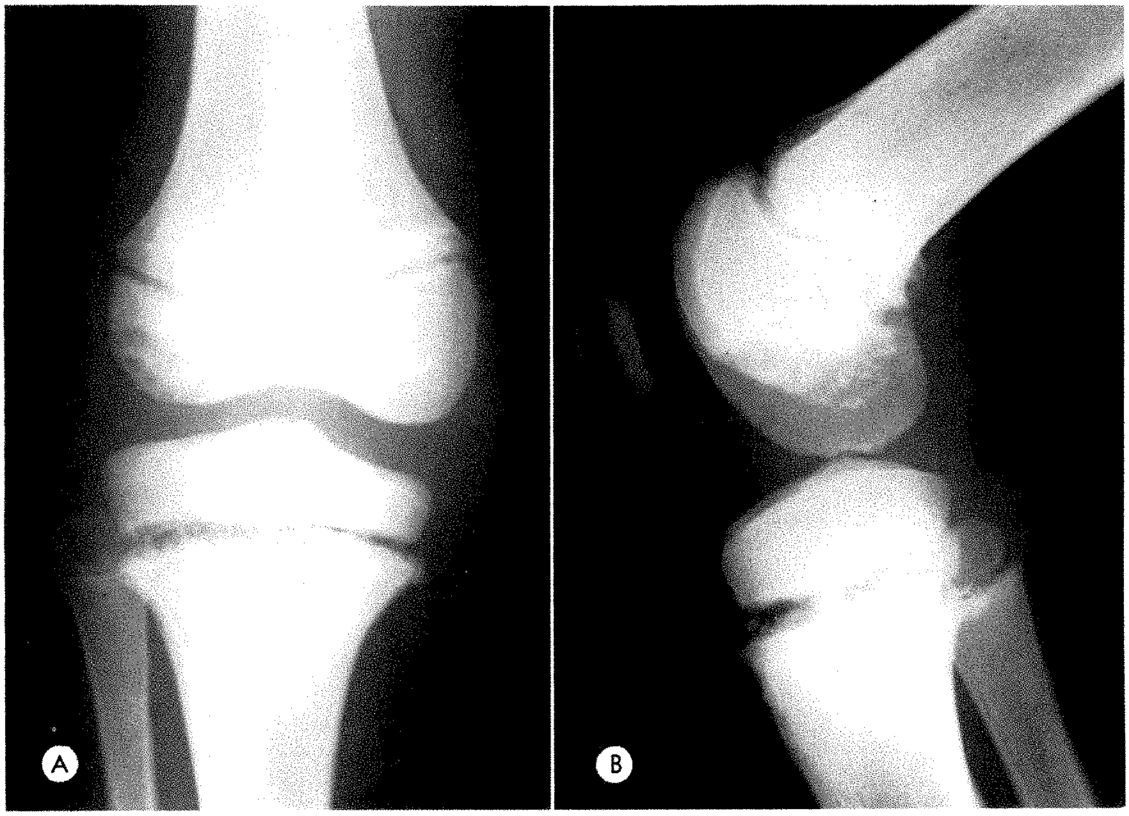

Fig. 3. *Right knee at age of 9 years.* (*A* and *B*) Sclerosis about the widened epiphyseal plates. Progression of aseptic necrosis of lateral femoral condyle.

CLINICAL FEATURES

Congenital indifference to pain is a disorder characterized by an intact peripheral sensory apparatus. All sensory stimuli can be identified at normal threshold levels.[3] Those with this disorder, however, do not respond in the usual manner to noxious stimuli. Burns and penetrating trauma are perceived as such but there is an absence of crying, discomfort, or defensive reaction as if the individual were indifferent. Some advocate the term "congenital insensitivity to pain" and believe that although these individuals possess the ability to discriminate sharp from dull, they accomplish this by means of a combination of touch and pressure clues and are actually insensitive to pain stimuli.[7] Between 1940 and 1960 there were 10 cases reported under the name of "congenital insensitivity to pain," and 13 cases under the name of "congenital indifference to pain."[7]

The manifestations are usually detected in the first years of life. Clinical histories include ulcers of the tongue, numerous bruises and injuries, burns from radiators and stoves, and neglected fractures of the extremities.

ROENTGEN FEATURES

The cardinal roentgen findings reflect the reaction of the skeletal system to repeated episodes of trauma at various stages of skeletal development. Fanconi and Ferrazzini[5] presented an excellent study of the skeletal features. Important contributions have also been made by Silverman and Gilden,[11] Murray,[8] and Caffey.[2] In Table I we have summarized the roentgen manifestations of this disorder.

The age of the individual determines the type of skeletal reaction. The periosteum in the infant is more loosely attached than in the older child or adult. Osseous injuries

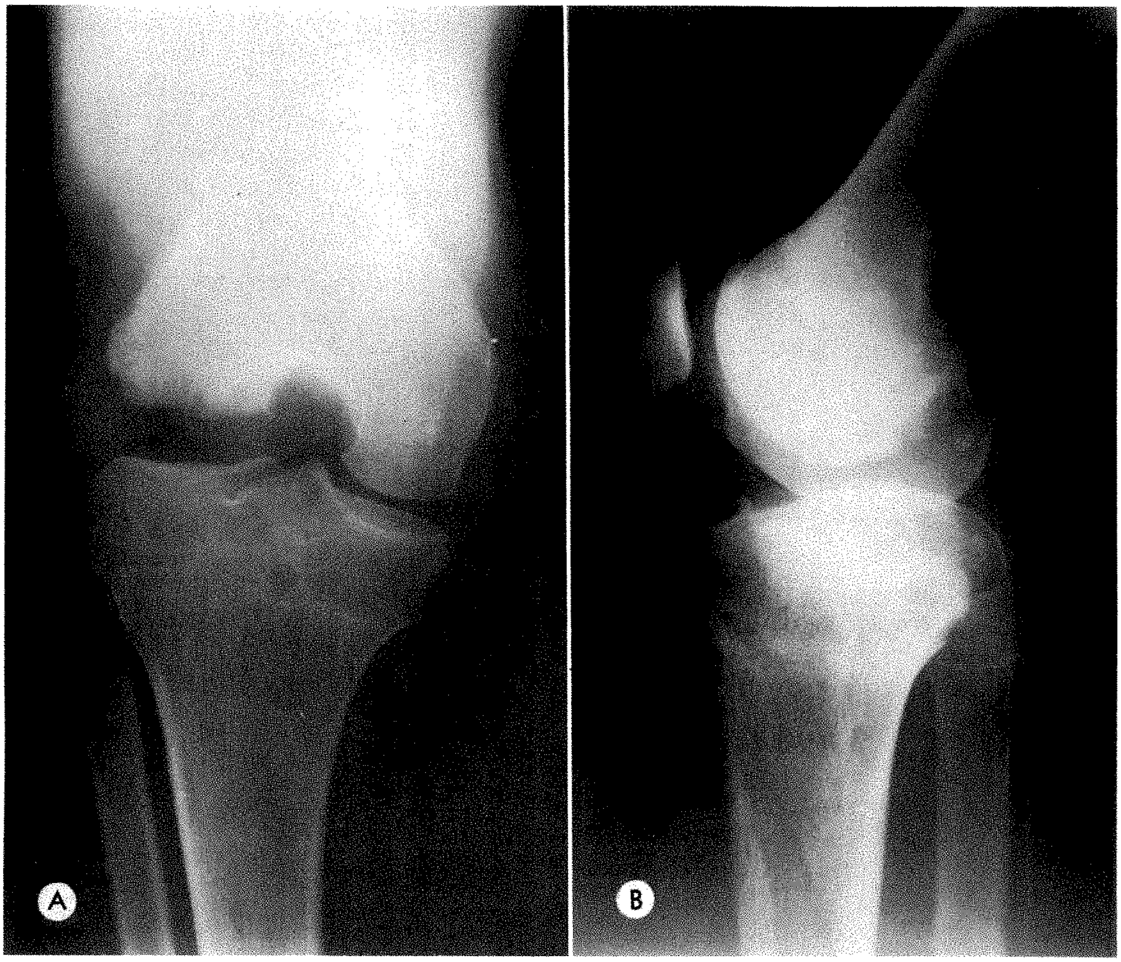

Fig. 4. *Right knee at age of 14 years.* (*A* and *B*) Advanced avascular necrosis of lateral femoral condyle.

in infants are associated with subperiosteal hemorrhage and eventually with an extensive participation by the periosteum in the repair process. Cortical thickening is a common phenomenon following osseous trauma in infancy.

In approximately one-third of the analyzed cases, ordinary fractures are encountered.[5] Thus, fractures of the skull, the long bones and the tubular bones of the hands and feet have been described. Abnormalities in the metaphyseal area as contrasted to the shafts of bone are typically encountered as a response to trauma in the growing skeleton. The provisional zone of ossification with the attached epiphysis may be disrupted and displaced. This changes the axis of growth of the shaft and produces a gross deformity. Following repeated microtrauma, one may encounter widening of the epiphyseal plates without actual displacement of the epiphysis (Fig. 3, *A* and *B*). Rickets has been erroneously diagnosed in such cases and the roentgenographic resemblance is no doubt present. Silverman and Gilden[11] cited the widening of the epiphyseal plates and theorized that the combination of metaphyseal abnormalities may be the childhood counterpart of the neuropathic joint of the adolescent and adult.

A most interesting feature of repeated skeletal microtrauma is the occurrence of areas of aseptic necrosis and osteochondritis in the juxta-articular regions of the weight-bearing long bones. The hip, the knee, and

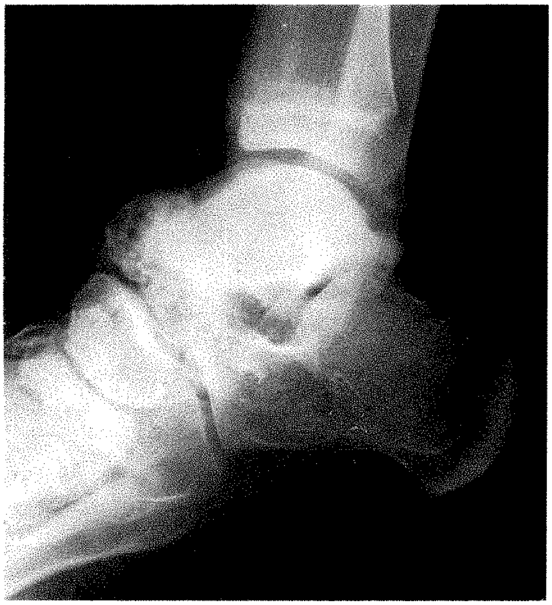

FIG. 5. *Right ankle at age of 14 years.* Right talonavicular joint with neuropathic change.

the ankle are most often affected. Repeated malposition from movement of insensitive joints beyond their normal range with associated stress on articular surfaces is the dynamic explanation for this occurrence. Prolonged joint stress may eventually lead to the disruption of joint integrity. The right knee of the patient under discussion with subluxation, joint effusion, synovial hypertrophy, degenerated cartilages, and loose osseous fragments indeed represents a neuropathic joint.[6] Other instances of neuropathic joints have been reported,[1,9] and Murray[8] indicates that one should suspect congenital indifference to pain in any child presenting with neuropathic joints.

A final general category of disease is that of inflammation. This is usually a subacute metaphyseal form of osteomyelitis. Most frequently, one encounters indolent areas of osteomyelitis presenting as fairly-well circumscribed metaphyseal foci.[1] The exact pathogenesis for the occurrence of osteomyelitis has not been definitively established. It is probably a result of a combination of neglected skin infections, infected burns, and trauma, the latter making for the localization of the inflammation. As the individual grows older, there is a tendency toward greater awareness of the disorder with compensation through avoidance of injury. Thus, gross fractures and active osteomyelitis are much less common in later childhood or adult life.

SUMMARY

A patient with congenital indifference to pain has been followed for 15 years. We

TABLE I

Cause	Skeletal Manifestations		
	Infancy	Early Childhood	Later Childhood and Adult Life
1. Macrotrauma	Metaphyseal Fragmentation	Frank Fractures	Fractures Relatively Uncommon
2. Repeated Microtrauma	Cortical Thickening	Aseptic Necrosis and Osteochondritis Widening of the Epiphyseal Plate	Neuropathic Joints Aseptic Necrosis and Osteochondritis
3. Infection	Uncommon	Chronic Osteomyelitis with Metaphyseal Predilection	Osteomyelitis Less Common

have attempted to explain and emphasize the following 4 characteristic roentgenologic features: (1) circumscribed metaphyseal foci of osteomyelitis; (2) widening of the epiphyseal plates; (3) multiple foci of aseptic necrosis involving the weight-bearing joints; and (4) neuropathic joints.

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TEETH IN EXTRAOVARIAN TERATOMAS

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BECAUSE teeth so rarely are found in extraovarian teratomas, a new case merits a short communication. Despite a rather thorough search of the literature, we have been able to find only a few well documented cases (Table I). In these instances, the teratomas have been found in the pituitary region,[18] pineal area,[3,6,12] third ventricle,[4] sacrococcygeal area,[8,16,17,19] anterior mediastinum,[13,20] posterior mediastinum,[10] stomach,[5,9] face,[7] and external auditory canal.[2,14]

TABLE I

Authors	Year	Type, Size and Location	Teeth	Sex of Patient	Age of Patient (yr.)
Boehm[4]	1919	Cystic, 3rd ventricle filled out and distended	Embryonal but no formed teeth	M	9
Becco[2]	1929	Auditory canal	Cyst with molar tooth	M	15
Rigby[18]	1932	Pituitary area	Numerous permanent teeth	M	2
Willis[20]	1935	Partly cystic, anterior mediastinum, 11×10×10 cm.	1 partially developed tooth	?	?
Raven[16]	1935	Cystic, rectal-sacrococcygeal area, 5×4×4 cm.	2 well formed teeth 1 ?	F	39
Marshall[14]	1936	External auditory canal	Dermoid cyst containing 2 imperfect molars (3rd molar missing)	?	15
Bochner and Scarff[3]	1938	Cystic, 3rd ventricle, 5.5 ×3.5×5.5 cm.	Several embryonal and formed teeth	M	9
Seeman[19]	1948	Cystic, sacrococcygeal area, 29×21×10 cm.	12 well formed teeth	M	29
Gross *et al.*[8]	1951	Sacrococcygeal area	Histologic evidence of dental tissue	Not given	Not given
Ravitch and Smith[17]	1951	Sacrococcygeal area	Histologic evidence of dental tissue	Not given	Not given
Erich and Johnsen[7]	1953	Right cheek	Multiple teeth	F	Congenital
Handelsman *et al.*[9]	1955	Mass, 12×8×6 cm. arising from anterior wall of stomach	Histologic evidence of dental tissue	M	4 mo.
Davidoff and Epstein[6]	1955	Pineal area	Well formed teeth	M	29
Le Roux[13]	1960	Anterior mediastinal mass	Present—not described	F	16
	1960	Anterior mediastinal mass connected to thymus by pedicle	Present—not described	M	38
Howard *et al.*[10]	1961	Posterior mediastinum, 5 cystic masses	Several teeth	M	4
Kroening[12]	1962	Cystic, pineal area, 6 cm. in diameter	1 bizarre tooth	M	3
Gorlin and Wolfson		Cystic, pineal area, 4 cm. in diameter	12 to 14 well formed teeth	M	12

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The so-called "teratoma of the pituitary" described by Pflüger and Schürmann[15] appears more likely to have been an ameloblastic odontoma or compound odontoma than a teratoma. A similar lesion was described by Kalnins and Rossi.[11] Beck,[1] in 1883, reported a pituitary teratoma with teeth, but the microscopic description would suggest a compound odontoma since other than dental tissues were absent. Because the anterior pituitary has its origin in oral ectoderm, the development of an odontogenic tumor in the pituitary area is certainly plausible.

REPORT OF A CASE

R.H., a 12 year old boy, was seen at the University of Minnesota Hospitals for the first time on November 8, 1951, complaining of double vision and an unsteady right hand. Five months before, he was unconscious for 3 days, having been thrown to the ground in a tractor accident. After the accident he noted double vision and was unable to move his right arm for a period of 1 month. Function of this extremity was, to a great extent, restored. One month before admission, the patient suffered a convulsion, which was followed by a second seizure 3 weeks later. The first attack was preceded by vertigo and followed by twitching of the right arm and head and 20 minutes of unconsciousness.

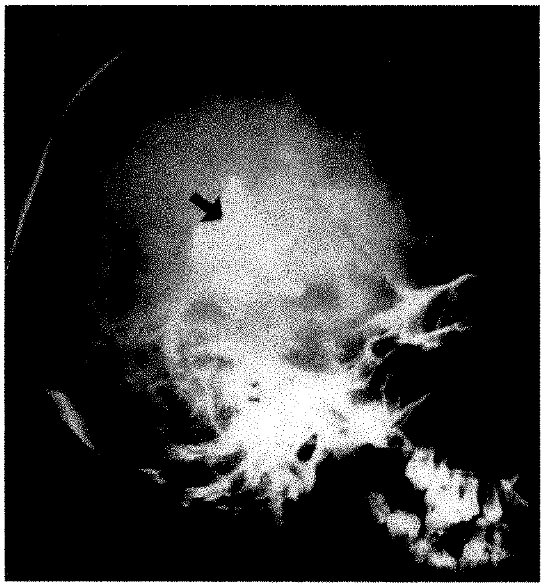

Fig. 1. Lateral roentgenogram demonstrating numerous teeth in various stages of development in the pineal area.

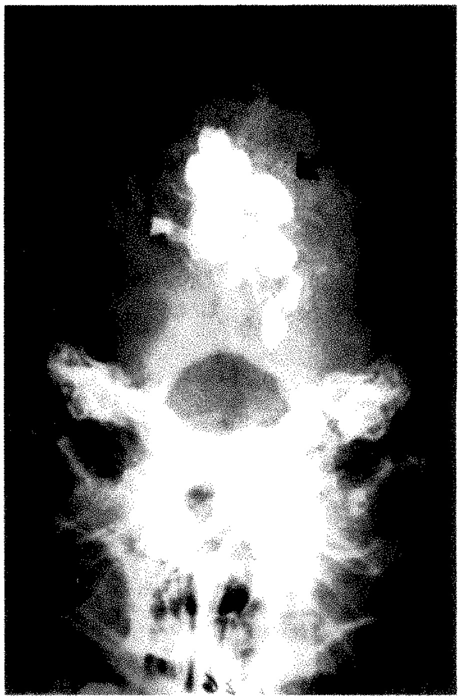

Fig. 2. Occipital or Towne view clearly showing the various crown forms of the teeth. The root development is largely missing.

Physical examination revealed the following pertinent findings: hyperactivity of reflexes (Babinski, knee jerk, biceps) on the right, tremors in all 4 extremities, sluggish pupillary reaction, nystagmus or lateral and upward gaze, and outward strabismus. Speech was slow, thick and labored. Intracranial pressure was not elevated.

Roentgenograms of the skull showed a mass with about 12 to 14 teeth in various stages of development in the pineal region (Fig. 1 and 2). The mass measured approximately 4×4×5 cm. On December 3, 1951, a craniotomy was carried out. Total extirpation of the tumor was not attempted. Only a single molar tooth and a portion of the wall of the cystic mass were removed. Histologic examination showed portions of bone, hair, stratified squamous epithelium and sebaceous glands.

The postoperative course was uneventful. A follow-up study was done several months later. The patient reported having mild headaches but no further convulsions and the mass had not increased in size.

SUMMARY

An unusual case of a tumor in the pineal region containing 12 to 14 teeth is reported.

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THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

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Representative on the Board of Chancellors of the American College of Radiology: Charles G. Stetson.

Forty-eighth Annual Meeting—Golden Anniversary of the American Radium Society: Camelback Inn, Phoenix, Ariz., April 13–16, 1966.

EDITORIAL

PEDIATRIC RADIOLOGY COMES OF AGE

INFANCY, childhood, adolescence, and maturity are recognized developmental stages of man. This arbitrary division can also be applied to organizations. The adult is appraised of his maturity by his action and interaction with his fellow man. When accepted by his associates, he is ready to take his place in society. In a similar manner, when organizations or groups have made a significant niche among their own associates, they also, so-to-speak, become mature. Their contributions are considered and accepted in their field. It is at this stage where the subspecialty of Pediatric Radiology is today.

Pediatric Radiology was in its infancy when John Caffey, in 1956 in this JOURNAL, reviewed the chronologic events to that date; it was indeed a vigorous infant. In 1958, there was a sufficient number of individuals interested in, and solely practicing, Pediatric Radiology—a fact which propelled the foundation of the Society of Pediatric Radiology. This Society added further impetus to the exchange of ideas and provided for a healthy childhood growth. The JOURNAL devoted several issues entirely to the problems of diagnostic pediatric roentgenology, furnishing undeniable proof that we have made a significant impact on our fellow radiological associates. Now in 1966, the May issue is again devoted in its entirety to this growing field. To be honored periodically by this distinguished JOURNAL is certainly an indication that the subspecialty of Pediatric Radiology has reached full maturity.

Some of the articles are written by individuals whose primary interests are not in pediatrics, whereas others are written by those who work exclusively in pediatrics. We who devote all of our efforts to Pediatric Radiology welcome those who find that they are stimulated by the untapped areas for investigation and feel obligated to contribute to the literature.

The steadily growing interest in childhood problems prompts the question of the availability of formal training. Some may even question whether the development of a subspecialty should be encouraged. Are we not subspecialized enough? Do not the principles and concepts pertaining to adult radiology also apply to children? To this latter question the answer is an unqualified no. It must be recognized that children are not just small adults. The pediatrician, during his early development, encountered the same type of opposition; today the role of the pediatrician in medicine is unquestioned.

Pediatric radiology is an amalgamation of pediatrics and radiology. The individual who chooses this field must have a genuine interest and a thorough knowledge of the child, as well as the probing stimulus of diagnostic radiology. It is obvious that training in this subspecialty must be obtained in institutions which devote all of their time and efforts to pediatrics; also, where the Radiology Department serves pediatricians exclusively.

Subspecialization has many precedents in adult medicine. This diversification has spread into the field of pediatrics. So vast has become the knowledge in dealing with children that at our own hospital in the Pediatric Department there are pediatricians whose total interests are confined to such areas as endocrinology, virology, hematology, neurology, cardiology, biochemistry, etc. The pediatric surgeon has also found it necessary to divide his interest so that we now have the pediatric neurosurgeon, the orthopod, and the urologist. Explosion of knowledge and challenges in these areas entice young men to these careers. Radiology is no different. Our own specialty, I feel certain, will also undergo further subspecialization. There is already evidence of this; *i.e.*, the neuroradiologist, the cardiovascular radiologist, etc.

The concept of radiology concentrated on the child as a full time occupation is not limited to the American Continent. In 1963, the European Society of Pediatric Radiology was formed. Successful Annual Meetings have been held since that time.

The dilemma in obtaining training for the pediatric radiologist is whether to study pediatrics first and follow with radiological training, or become a radiologist first and study pediatrics later. Many of today's recognized pediatric radiologists developed either as pediatricians, later becoming radiologists or *vice versa*. The radiologist who devoted his time in a pediatric hospital learned by so-called on-the-job-training. It was necessary for him to grasp pediatrics while practicing radiology. At the present time, I believe this type of experience is unwarranted, since methods of obtaining a formal training are available.

A young man, who early in his career has decided to become a pediatric radiologist, should first obtain his resident training in diagnostic radiology with the primary aim of obtaining Board certification in diagnosis only. Two to three years in the adult diagnostic department would give the early trainee a basic foundation in the concepts of radiological techniques, etc., as it is applied to medicine. Ultimately, at least 2 years in a Pediatric Radiology Department would be almost mandatory. Board certification could then be obtained in diagnostic roentgenology as the requirements would be fulfilled. It is hoped that in 2 years in a Pediatric Radiology Department, a sufficient knowledge of pediatrics would be obtained, this being a necessary adjunct to future practice. In line with this, The Children's Bureau of the U.S.P.H.S. is sponsoring a 2 year training Fellowship in Pediatric Radiology at a major institution in this country.

It is well recognized that any proper training in pediatrics prior to embarking upon a radiological career is to the advantage of the individual. Those possessing Board certification in pediatrics and radiology have an ideal arrangement but this is not mandatory. Training in this manner is usually long.

The question now arises as to whether there should be a Board examination in Pediatric Radiology. At this time, I think it would be unnecessary. An individual who has obtained his certificate in radiology should be qualified if he has served a sufficient amount of time in a well recognized institution under a pediatric radiologist. His motivation and exposure in this type of environment should be adequate. Another examination would not be necessary. Presently we have a fine corps of radiologists, none of whom has any Board certification in this subspecialty. A few have obtained recognition starting out as pediatricians with subsequent radiology training and do not have Board certification in radiology. Examination does not necessarily make one qualified. Most of the young men today in this field are well trained both in radiology and pediatrics.

What then is the future for the young man embarking on this career? Undoubtedly, it is bright. There is an explosive amount of knowledge in all phases of pediatrics and with it diagnostic radiology will increase in importance. It represents an exciting challenge to the young man. Many areas are as yet unexplored. One of the most challenging is that of the premature and newborn. Our subspecialty of Pediatric Radiology is established. It has reached full maturity. It is for us to stimulate it, nourish it, and keep it dynamically progressive.

HARVEY WHITE, M.D.
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INTERNATIONAL COMMISSION ON RADIOLOGICAL UNITS AND MEASUREMENTS (ICRU)

REPORT TO THE INTERNATIONAL EXECUTIVE COMMITTEE OF THE ELEVENTH INTERNATIONAL CONGRESS OF RADIOLOGY, ROME, 1965

PREVIOUS ACTIVITIES

BEFORE turning to the actual program which the ICRU has been engaged in since the Tenth International Congress of Radiology, it would seem appropriate to mention some of the results of the work accomplished prior to the Tenth Congress. As was reported to the International Executive Committee in 1962, ICRU efforts between 1959 and 1962 resulted in the publication of 6 major reports:

ICRU Report 10a on Radiation Quantities and Units (NBS Hdb. 84)
ICRU Report 10b on Physical Aspects of Irradiation (NBS Hdb. 85)
ICRU Report 10c on Radioactivity (NBS Hdb. 86)
ICRU Report 10d on Clinical Dosimetry (NBS Hdb. 87)
ICRU Report 10e on Radiobiological Dosimetry (NBS Hdb. 88)
ICRU Report 10f on Methods of Evaluating Radiological Equipment and Materials (NBS Hdb. 89).

DEVELOPMENT OF NEW SUBSTRUCTURE AND MODE OF OPERATION

Since this last report in August, 1962, the ICRU has devoted itself principally to the elaboration of the new substructure and mode of operation adopted by the Commission in 1962, and to the utilization of this new substructure for the initiation of new projects. The Commission defined 3 broad areas of concern to the ICRU—Activity, Radiation, and Problems of Joint Interest to the ICRU and the International Commission on Radiological Protection (ICRP). The ICRU divided these 3 major areas into eight sub-areas with which the Commission expects to be primarily concerned during the next few years. The division of work agreed upon is as follows:

I. Activity
    A. Fundamental Physical Parameters and Measurement Techniques
    B. Medical and Biological Applications
II. Radiation
    A. Fundamental Physical Parameters
    B. X Rays, Gamma Rays and Electrons
    C. Neutrons and Heavy Particles
    D. Medical and Biological Applications (Therapy)
    E. Medical and Biological Applications (Diagnosis)
III. Problems of Joint Interest to the ICRU and the ICRP
    A. Radiation Protection Instrumentation and Its Application

The Commission has established 8 Planning Boards to guide ICRU activities in each of the sub-areas and named Commission Sponsors to provide liaison between the Commission and the Planning Boards. The constitution of the Planning Boards is now essentially complete and their members and Commission Sponsors are listed in Annex 1 of this report.

The Planning Boards began studies of the needs in each of the sub-areas; and proposed, and received Commission approval, for programs to be undertaken by Task Groups working under the guidance of the Planning Boards. This process has thus far resulted in the establishment of the following Task Groups:

Under the *Planning Board on Activity—Fundamental Physical Parameters and Measurement Techniques:*
  Task Group 1. Measurement of Low-Level Radioactivity
  Task Group 2. Specification of Accuracy in Certificates of Activity of Sources for Calibration Purposes
  Task Group 3. Specification of High Activity Gamma Ray Sources

Under the *Planning Board on Activity—Medical and Biological Applications:*
  Task Group 1. *In Vivo* Measurements of Radioactivity
  Task Group 2. Scanning
  Task Group 3. Tracer Kinetics
  Task Group 4. Methods of Assessment of Dose in Tracer Investigations
Under the *Planning Board on Radiation—X Rays, Gamma Rays and Electrons:*
  Task Group 1. Radiation Dosimetry; X Rays from 5 to 150 kV
  Task Group 2. Radiation Dosimetry; X and Gamma Rays from 0.6 to 100 MV
  Task Group 3. Electron Beam Dosimetry
Under the *Planning Board on Radiation—Heavy Particles:*
  Task Group 1. Dose as a Function of LET
  Task Group 2. High Energy and Space Radiation Dosimetry
Under the *Planning Board on Radiation—Neutron, Fluence and Kerma:*
  Task Group 1. Neutron Fluence, Energy Fluence, Neutron Spectra and Kerma
Under the *Planning Board on Radiation—Medical and Biological Applications (Therapy):*
  Task Group 1. Measurement of Absorbed Dose at a Point in a Standard Phantom (Absorbed Dose Determination)
  Task Group 2. Methods of Arriving at the Absorbed Dose at any Point in a Patient (*In Vivo* Dosimetry)
  Task Group 3. Methods of Compensating for Body Shape and Inhomogeneity and of Beam Modification for Special Purposes (Beam Modification)
  Task Group 4. Statement of the Dose Achieved (Dosage Specification)
Under the *Planning Board on Radiation—Medical and Biological Applications (Diagnosis):*
  Task Group 1. Photographic Materials and Screens
  Task Group 2. Cine Systems
  Task Group 3. TV Systems
Under the *Planning Board on Radiation Protection Instrumentation and Its Application:*
  Task Group 1. Radiation Protection Instrumentation Handbook—Part I
  Task Group 2. Neutron Instrumentation and Its Application to Radiation Protection

The titles of these Task Groups give an indication of the breadth of the program now being undertaken by the ICRU.

The selection of personnel for the Task Groups is now largely completed and, indeed, many of them have already begun their work.

ACTIVITIES OF THE PLANNING BOARDS AND TASK GROUPS

The belief that the new substructure and mode of operation will facilitate the work of the Commission is supported by the experience of the last 2½ years. The Planning Boards have actively undertaken the precise definition of the work that needs to be done, and have stipulated the scope of activity for each of the Task Groups thus far established. Many of these Task Groups have held initial meetings and in some cases preliminary drafts of proposed ICRU reports have already been prepared.

The Commission and the Chairmen of the Planning Boards met in Rome from October 28, 1963 through November 1, 1963. This meeting was primarily concerned with Commission review of the efforts which the Planning Boards were initiating or proposing to undertake. Many Planning Board meetings have been held since 1962, both before and after the 1963 meeting of the Commission.

ICRU RELATIONSHIPS WITH OTHER ORGANIZATIONS

One of the features of ICRU activity during the last 3 years has been the development of relationships with other organizations interested in problems of radiation quantities, units and measurements. In addition to its close relationship with the ICRP, and its financial relationships with the International Society of Radiology, the World Health Organization and the International Atomic Energy Agency, the ICRU has also developed relationships of varying intensity with several other organizations. The Commission has a formal relationship with the United Nations Scientific Committee on the Effects of Atomic Radiation, whereby ICRU observers are invited to attend UNSCEAR meetings. The Commission informally exchanges with the International Standards Organization notifications of meetings and is formally designated for liaison with two of the ISO Technical Committees.

The ICRU also corresponds and exchanges final reports with the following organizations: International Labor Organization; Food and Agriculture Organization of the United Nations; Council for International Organization of Medical Sciences; International Council of Scientific Unions; Bureau International des Poids et Mesures; United Nations Educational, Scientific and Cultural Organization; International Electrotechnical Commission; and the International Union of Pure and Applied Physics.

In connection with its relationship with the ICRP, each of the Commissions in 1962, determined to foster closer coordination of the programs of the Commissions and in implementation of this determination directed the officers to develop means for such coordination based on frequent meetings between the officers and extensive exchange of documents. This directive resulted in meetings of the officers of the ICRP and ICRU in Paris in May 1963 and in London in September 1964. These meetings permitted an exchange of information which not only contributed to the coordination of future meetings and activities of the two Commissions, but also enabled the secretariats to develop means for cooperation in the use of unique resources available to each.

COMMISSION FINANCES

As indicated above, the ICRU has financial relationships with the International Society of Radiology (ISR), the World Health Organization (WHO), and the International Atomic Energy Agency (IAEA). In October 1962, the ISR contributed $5,000 to cover the period until the Eleventh International Congress of Radiology. The agreement between the ICRU and the WHO whereby the WHO makes available $4,000 per year has been continued and it is expected that it will remain in force for some time to come. Similarly, the Commission and the IAEA have maintained in force a series of agreements whereby the Agency makes available to the ICRU $6,000 per year. It is expected that this arrangement will also be continued. Finally, the importance of the Ford Foundation grant received by the ICRU in 1960 must be emphasized. The funds from this grant, $37,000 per year, have proved of substantial significance in permitting the Commission to move forward quickly in its efforts to meet the expanded needs for its work.

The Commission wishes to express its deep appreciation to all of the organizations mentioned above and to others that have contributed so importantly to its work.

MEMBERSHIP

The Commission has elected the following individuals to Commission membership for the period between the Eleventh and Twelfth International Congress of Radiology:

Elected Senior Advisors: F. Ellis, member since 1950; H. Fränz, member since 1959; V. A. Petrov, member since 1962.

New Members 1965–1969: F. P. Cowan (U.S.A.); J. F. Fowler (United Kingdom); and R. H. Morgan (U.S.A.).

Continuing Members 1965–1969: A. Allisy (France); J. W. Boag (United Kingdom); R. H. Chamberlain (U.S.A.); F. Gauwerky (Germany); H. E. Johns (Canada); K. Lidén (Sweden); H. H. Rossi (U.S.A.); L. S. Taylor (U.S.A.); M. Tubiana (France); and H. O. Wyckoff (U.S.A.).

Commission Officers 1965–1969: L. S. Taylor, *Chairman;* M. Tubiana, *Vice-Chairman;* H. O. Wyckoff, *Secretary;* W. R. Ney, *Technical Secretary* (not a member of the Commission).

ICRU RULES

The Commission wishes to propose a change in its rules to make a provision for the establishment of an ICRU medal honoring its late Vice-Chairman, Dr. L. H. Gray.

Lauriston S. Taylor

Annex I

COMMISSION SPONSORS AND MEMBERS OF THE PLANNING BOARDS

Planning Board on Activity—Fundamental Physical Parameters and Measurement Techniques: K. Lidén, *Sponsor;* F. W. Spiers, Chairman; H. Houtermans; W. B. Mann; J. K. Miettinen.

Planning Board on Activity—Medical and Biological Applications: M. Tubiana, *Sponsor;* H. Vetter, Chairman; C. Kellershohn; K. E. Scheer; N. Trott.

Planning Board on Radiation—Fundamental Physical Parameters: J. W. Boag, *Sponsor.* (No further elaboration of this Planning Board's structure is planned at present, but the Board will function on an *ad hoc* basis as necessary.)

Planning Board on Radiation—X Rays, Gamma Rays and Electrons: A. Allisy, *Sponsor;* W. Pohlit, Chairman; D. V. Cormack; C. Garrett; A. Somerwil.

Planning Board on Radiation—Heavy Particles: H. H. Rossi, *Sponsor;* W. K. Sinclair, Chairman; F. H. Attix; T. Brustad; B. Larsson.

Planning Board on Radiation—Neutron, Fluence and Kerma: J. F. Fowler, *Sponsor;* A. H. W. Aten, Jr., Chairman; R. S. Caswell; K. W. Geiger; F. Netter.

Planning Board on Radiation—Medical and Biological Applications (Therapy): H. E. Johns, *Sponsor;* F. Gauwerky, *Co-sponsor;* W. J. Meredith, Chairman; F. Ellis, Senior Advisor; J. Dutreix.

Planning Board on Radiation—Medical and Biological Applications (Diagnosis): R. H. Chamberlain, *Sponsor;* R. H. Morgan, *Co-sponsor;* O. Olsson, Chairman; E. D. Trout, Vice-Chairman; E. Zieler; R. D. Moseley; J. J. Stevenson; T. A. Tristan.

Planning Board on Radiation Protection Instrumentation and Its Application: H. O. Wyckoff, *Sponsor;* F. P. Cowan, *Co-sponsor;* L. Larsson, Chairman; C. Garrett; E. E. Smith.

NEWS ITEMS

X RAY EXHIBIT AT HARVARD UNIVERSITY

One of the most comprehensive exhibits yet assembled on the early history and later development of the X ray in the United States comprises the initial display in the Francis A. Countway Library of Medicine in the Harvard Medical Area, Boston, Massachusetts.

The materials included in the exhibit were brought together by Dr. Lloyd E. Hawes of Dedham, Massachusetts, Assistant in Radiology at the Harvard Medical School and Honorary Curator of the Historical Collection in Radiology at the Countway Library.

The exhibit is arranged chronologically in eight cases. The discoverer of the X ray, Dr. Wilhelm K. Röntgen of Germany, is well represented by his famous 1895 X ray of his wife's hand; by an outline of courses given by him at Würzburg in 1896; by a series of letters; and by 3 early articles on the X ray.

Selections from books, in a special collection of X ray and related science material, are arranged in chronologic order.

Among the important manuscripts on display are letters by Madame and Pierre Curie, Dalton, Tyndall, Crookes, Helmholtz, Ampère, Volta, Gauss, and other leading figures relating to the science, as well as selections from the works of Dr. George W. Holmes along with his portrait.

The variety of X ray equipment includes a collection of tubes used by the first clinical roentgenologist in the United States, Dr. Francis Williams of Boston. The meticulous notebook of Dr. Walter B. Cannon and his electrostatic generator, as restored by William Faxon, president of Comstock and Wescott Company, Cambridge, are also displayed.

Standing free in the center of the exhibit is a Van de Graaff 2 million volt generator which was used in X ray treatment at the Philadelphia Oncologic Hospital from 1942 to 1965. It is flanked by two models; the first million volt Van de Graaff generator used at Harvard's Huntington Memorial Cancer Hospital, and the first Van de Graaff generator used at the Massachusetts General Hospital. Of particular interest are 2 glass door knobs, one of which was on the inside of the X ray treatment room and was turned a deep purple by radiation, and the other which was located on the outside of the lead shielded door and remained perfectly clear.

BIANNUAL GLACIER PARK RADIOLOGIC-ORTHOPEDIC SEMINAR

The Biannual Glacier Park Radiologic-Orthopedic Seminar will be held July 20, 21 and 22, 1966, at Glacier Park Lodge, East Glacier, Montana.

The following topics will be discussed: July 20, benign and malignant bone tumors; July 21, congenital abnormalities of extremities; and July 22, difficult bone trauma problems.

Guest Speakers will include: David Pugh, M.D., Mayo Clinic; William R. Christensen, M.D., University of Utah; Sherman Coleman, M.D., University of Utah; and William Bailey, M.D., University of Michigan.

For reservations, please write to E. J. Drouillard, M.D., Box 1132, Missoula, Montana 59801.

VII CONGRESO DE ELECTRORADIÓLOGOS DE CULTURA LATINA—I CONGRESO DE LA ASOCIACIÓN EUROPEA DE RADIOLOGÍA

The Federation of Electroradiologic Physicians of Latin Culture will hold its Seventh Congress in Barcelona, April 2–8, 1967. At the same time, the European Association of Radiology will inaugurate its First Congress.

Dr. Don Francisco Gálvez Aremengaud was elected President of the united Congress.

The office of the Secretary General is at the Faculty of Medicine of the City of Barcelona.

An excellent program is being prepared. For further information please write to Dr. F. Manchón, Secretario General, Facultad de Medicina, Barcelona, Spain.

SHORT-TERM CLINICAL TRAINEESHIP PROGRAM IN THE DIAGNOSIS AND TREATMENT OF CANCER

The American Cancer Society announces a program of short-term (3 to 6 months) clinical traineeships in the diagnosis and treatment of cancer for residents and Board certified or qualified clinicians.

The purpose is to provide training or experience in special techniques in cancer diagnosis or treatment which are not available at the physician's own institution or which he did not receive in earlier training. Traineeships are available only in departments holding regular Clinical or Advanced Clinical Fellowships of the American Cancer Society for the period for which training is requested. (Exceptions may be made in certain instances.)

The candidate for this program must be a citizen of the United States, a graduate of a medical school in the United States, Puerto Rico or Canada and under the age of 41. The stipend is $300 per month and may be supplemented by the institution or other sources except not directly from private practice.

Applications must be submitted by Chiefs of Services and Chairmen of Departments, not by the individual applicant.

For further information please write to the Director of Professional Education, American Cancer Society, Inc., 219 East 42nd Street, New York, New York 10017.

AMERICAN SOCIETY FOR DIAGNOSTIC ULTRASOUND

This Society was founded during the International Conference on Diagnostic Ultrasound in Pittsburgh, Pennsylvania. The purpose of the Society is to foster the science of experimental and clinical diagnostic ultrasonics; to disseminate information of advancements in this science as well as in ultrasonic instrumentation; and to promote high standards of clinical diagnostic ultrasonic procedures.

Officers of the Society are: *President*, Joseph H. Holmes, M.D., University of Colorado; *Vice-President*, Claude Joyner, M.D., University of Pennsylvania; *Treasurer*, Edward W. Purnell, M.D., Western Reserve University; and *Secretary*, Charles C. Grossman, M.D., University of Pittsburgh.

Further information may be obtained from the Secretary, Dr. Charles C. Grossman, 552 N. Neville Street, Pittsburgh, Pennsylvania 15213.

BOOK REVIEW

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

FOUNDATIONS OF ANESTHESIOLOGY. In two volumes. By Albert Faulconer, Jr., M.D., M.S. in Anesthesiology, Head, Section of Anesthesiology, Mayo Clinic; Professor of Anesthesiology, Mayo Graduate School of Medicine, University of Minnesota, Rochester, Minn.; and Thomas E. Keys, A.B., M.A., Librarian, Mayo Clinic; Associate Professor of History of Medicine, Mayo Graduate School of Medicine, University of Minnesota, Rochester, Minn. Cloth. Pp., Vol. I, 716, Vol. II, 621, both with some illustrations. Price, $38.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1965.

This book in two Volumes presents an organized collection of classic scientific articles which *are* the "Foundations of Anesthesiology."

The work will be found useful as a reference source to all and is a fine background for those young in Anesthesiology and allied diciplines. The printing is excellent. The life sketches of the many authors offer an intriguing interlude between the papers, marred a bit, perhaps, by frequent repetition of the lives of the "giants."

The ingenuity and perseverance of early scientists in fabricating their own equipment and producing their own chemicals should give modern researchers pause, and the high order of the guardian angels present when these men deliberately administered general anesthetics to each other in the face of known full stomachs, leaves one completely awed. Big, but very worthwhile.

A. B. STEARNS, M.D.

BOOKS RECEIVED

CURRENT PERSPECTIVES IN CANCER THERAPY. Edited by William S. Blakemore, M.D., Professor of Surgery, University of Pennsylvania School of Medicine; Chairman, Department of Surgery, University of Pennsylvania, Division of Graduate Medicine, School of Medicine, Philadelphia; and I. S. Ravdin, M.D., Professor of Surgery, University of Pennsylvania School of Medicine, and Vice President for Medical Affairs, University of Pennsylvania, Philadelphia. Cloth. Pp. 234, with 50 illustrations. Price, $8.50. Hoeber Medical Division, Harper & Row, Publishers, Inc., 49 East 33rd Street, New York, N. Y., 1966.

MEDICAL SPELLING GUIDE: A REFERENCE AID. Compiled by Carrie E. Johnson, R.N., R.R.L., B.S., Medical Record Librarian, St. Petersburg, Fla.; with a foreword by Doris E. Gleason, R.R.L., Director, Medical Record Research Bureau, Chicago, Ill. Cloth. Pp. 538. Price, $10.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1966.

ASPECTS OF VENOUS FUNCTION IN THE LOWER LIMBS. By John Ludbrook, B.Med.Sc., Ch.M. (N.Z.), F.R.C.S., F.R.A.C.S., Professor of Surgery, University of New South Wales; Honorary Surgeon, Prince Henry and Prince of Wales Hospitals, Sydney, Australia; Former Senior Lecturer in Surgery, University of Otago, Dunedin, New Zealand; Leverhulme Research Fellow, St. Mary's Hospital, Paddington, London, England. Cloth. Pp. 137, with some illustrations. Price, $6.75. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1966.

SURGICAL APPLICATIONS OF LASER. By Paul Edward McGuff, B.S., M.D., M.S. (Surg.), Ph.D. (Surg.), Director and Chief Surgeon, Laser Medical Research Foundation, Institute of Biological and Medical Sciences and Laser Tumor Institute, Boston, Mass.; Formerly, Assistant in Surgery, Tufts University School of Medicine Surgical Staff, New England Center Hospital, Boston Dispensary. Cloth. Pp. 200, with many illustrations. Price, $10.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1966.

THE YEAR BOOK OF RADIOLOGY: (1965–1966 Year Book Series). Radiologic Diagnosis edited by John Floyd Holt, M.D., Professor, Department of Radiology, University of Michigan; and Walter M. Whitehouse, M.D., Professor and Chairman, Department of Radiology, University of Michigan. Radiation Therapy edited by Howard B. Latourette, M.D., Professor, Department of Radiology, University of Iowa. Cloth. Pp. 478, with 328 illustrations. Price, $12.00. Year Book Medical Publishers, Inc., 35 E. Wacker Drive, Chicago, Ill., 1966.

INTERNATIONAL SYMPOSIUM ON GENES AND CHROMOSOMES: STRUCTURE AND FUNCTION. Held at Buenos Aires, Argentina, Nov. 30–Dec. 4, 1964. National Cancer Institute Monograph 18, December, 1965. Edited by Professor Juan Il Valencia and Dr. Rhoda F. Grell. Cloth. Pp. 354, with many illustrations. Price, $3.50. U. S. Department of Health, Education, and Welfare, Public Health Service, National Cancer Institute, Bethesda, Md. For sale by the Superintendent of Documents, U. S. Government Printing Office, Washington, D. C., 1965.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. C. Allen Good, Mayo Clinic, Rochester, Minn. Annual meeting: San Francisco Hilton Hotel, San Francisco, Calif., Sept. 27–30, 1966.

American Radium Society
Secretary, Dr. John L. Pool, 444 East 68th Street, New York, N. Y. 10021. Annual meeting: Camelback Inn, Phoenix, Ariz., April 13–16, 1966 (Golden Anniversary).

Radiological Society of North America
Secretary-Treasurer, Dr. Maurice Doyle Frazer, 1744 South Fifty-eighth St., Lincoln, Neb. Annual meeting: Palmer House, Chicago, Ill., Nov. 27–Dec. 2, 1966.

American College of Radiology
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Ill. Annual meeting: Beverly Hilton Hotel, Los Angeles, Calif., Jan. 21–Feb. 4, 1967.

Section on Radiology, American Medical Association
Secretary, Dr. Clyde A. Stevenson, Sacred Heart Hospital, West 101 Eighth Ave., Spokane 4, Wash. Annual meeting: Chicago, Ill., June 26–30, 1966.

American Board of Radiology
Secretary, Dr. H. Dabney Kerr. Correspondence should be directed to Kahler Hotel Building, Rochester, Minn.

The Spring 1966 examination will be held at the Terrace Hilton Hotel, Cincinnati, Ohio, June 6–10, inclusive. The deadline for filing applications for this examination was December 31, 1965.

The Fall 1966 examination will be held at the Washington Hilton Hotel, Washington, D.C., December 5–9, inclusive. The deadline for filing applications is June 30, 1966.

American Association of Physicists in Medicine
Secretary, Leonard Stanton, Hahnemann Medical College, 230 N. Broad St., Philadelphia, Pa. 19102. Annual meeting to be announced.

American Club of Therapeutic Radiologists
Secretary, Dr. J. A. del Regato, Penrose Cancer Hospital, Colorado Springs, Colo.

Twelfth International Congress of Radiology
President, Dr. Kempo Tsukamoto, Chiba, Japan. Meeting: Tokyo, Japan, Sept. 1969.

Ninth Inter-American Congress of Radiology
Counselor for the United States, Dr. Philip J. Hodes, Jefferson Medical College Hospital, 11th and Walnut Streets, Philadelphia 7, Pennsylvania.
President, Dr. Leandro Zubiaurre, Montevideo, Uruguay. Meeting: Montevideo, Uruguay, 1967.

Inter-American College of Radiology
President, Dr. Oscar Soto, H. Urteaga 480, Lima, Perú.

Alabama Radiological Society
Secretary, Dr. Walter Brower, Birmingham, Ala. Meets time and place of Alabama State Medical Association.

American Nuclear Society
Treasurer, Raymond Maxson, 86 E. Randolph St., Chicago, Ill. Annual meeting to be announced.

American Society for Diagnostic Ultrasound
Secretary, Dr. Charles C. Grossman, 552 N. Neville St., Pittsburgh, Pa. 15213.

Arizona Radiological Society
Secretary-Treasurer, Dr. George Gentner, 3435 W. Durango, Phoenix, Ariz. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

Arkansas Chapter of American College of Radiology
Secretary-Treasurer, Dr. James R. Morrison, 550 S University, Little Rock, Ark. 72205.

Arkansas Radiological Society
Secretary, Dr. Charles W. Anderson, 1108½ Poplar, Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

Association of University Radiologists
Secretary-Treasurer, Dr. Morton M. Kligerman, Dept. of Radiology, Yale University Medical School, New Haven, Conn. Annual meeting: University of Arkansas, Little Rock, Ark., May 13–14, 1966.

Atlanta Radiological Society
Secretary, Dr. Donald R. Rooney, Burnt Hickory Road, Marietta, Ga. Meets monthly except during three summer months, on third Tuesday, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

Bavarian-American Radiologic Society
Secretary, Dr. Roy R. Deffebach, Major, MC, Radiology Service, 5th General Hospital, APO 154, New York, N. Y. Meets quarterly.

Blockley Radiological Society
Secretary-Treasurer, Dr. R. John Gould, 441 Lombardy Rd., Drexel Hill, Pa. 19026.

Bluegrass Radiological Society
Secretary-Treasurer, Dr. Arthur Lieber, University of Kentucky, University Hospital, Lexington, Kentucky. Meets quarterly.

Brooklyn Radiological Society
Secretary, Dr. Edward Feely, St. Johns Episcopal Hospital, 480 Herkimer St., Brooklyn, N. Y. Meets first Thursday of each month, October through June.

Buffalo Radiological Society
Secretary, Dr. Richard Munschauer, 130 Hodge Ave., Buffalo, N.Y. 14222. Meets second Monday evening each month, October to May inclusive.

California Radiological Society
Secretary, Dr. L. Henry Garland, Suite 1739, 450 Sutter St., San Francisco, Calif. Meets annually during meeting of California Medical Association.

Catawba Valley Radiological Society
Secretary, Dr. Emmett R. White, P. O. Box 303, Rutherford College, N. C. Meets every Tuesday, Dept. of Radiology, Valdese General Hosp., Valdese, N. C. at 12:00 P.M.

Central New York Radiological Society
Secretary-Treasurer, Dr. Robert A. Bornhurst, State Univ. Hospital, 750 E. Adams St., Syracuse, N. Y. 13210. Meets first Monday each month, October through May.

Central Ohio Radiological Society
Secretary-Treasurer, Dr. Atis K. Freimanis, Ohio State Univ. Hospitals, 410 W. 10th Ave., Columbus, Ohio 43210. Meets second Thursday in October, November, January, and March 15 and May 19 at Fort Hayes Hotel, Columbus, Ohio.

Central Society of Nuclear Medicine
Secretary, Dr. Robert S. Landauer, Radiation Center Bldg., 1903 West Harrison St., Chicago 12, Ill.

Chicago Roentgen Society
Secretary-Treasurer, Dr. Robert D. Moseley, Jr., Dept. of Radiology, Univ. of Chicago, 950 E. 59th St., Chicago 37, Ill. Meets second Thursday of each month, October to April except December at the Pick-Congress Hotel at 8:00 P.M.

Cleveland Radiological Society
Secretary-Treasurer, Dr. James Christie, 10515 Carnegie

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Avenue, Cleveland, Ohio. Meetings at 7:00 p.m. on fourth Monday of October, November, January, February, March and April.

Colorado Radiological Society

Secretary, Dr. George F. Wertz, 1801 High St., Denver, Colo. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society

Secretary, Dr. William W. Walthall, Jr., 130 Maple St., Springfield, Mass. Meets in April and October.

Dallas-Fort Worth Radiological Society

Secretary-Treasurer, John S. Alexander, 1217 W. Cannon, Ft. Worth, Tex. Meets monthly, third Monday, at Southwest International Airport at 6:30 p.m.

Detroit Roentgen Ray and Radium Society

Secretary, Dr. Robert L. Willis, Harper Hospital, Detroit 1, Mich. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 p.m.

East Bay Radiological Society

Secretary, Dr. Tom H. Piatt, 12 Camino Encinas, Orinda, Calif. 94563. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society

Secretary, Dr. C. H. Kimball, 2200 Harris Circle, Cleveland, Tenn. Meets in January and September.

Eastern Radiological Society

Secretary, Dr. James F. Martin, North Carolina Baptist Hospital, Winston-Salem, N. C.

Florida Radiological Society

Secretary, Dr. John C. Jowett, Orlando, Fla. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society

Secretary-Treasurer, Dr. Garth R. Drewry, Tampa General Hospital, Tampa 6, Fla. Meets in January, April, July and October.

Georgia Radiological Society

Secretary, Dr. I. R. Berger, 1010 Prince Ave., Athens, Ga. Meets in spring and fall with Annual State Society Meeting.

Greater Miami Radiological Society

Secretary-Treasurer, Dr. Arthur R. Miller, North Miami General Hospital, 1701 N.E. 127th St., North Miami, Fla. Meets monthly, third Wednesday at 8:00 p.m. at Jackson Memorial Hospital, Miami, Fla.

Greater St. Louis Society of Radiologists

Secretary-Treasurer, Dr. Mark D. Eagleton, 950 Francis Place, St. Louis, Mo.

Houston Radiological Society

Secretary, Dr. William A. Vint, 1004 Seymour, Pasadena, Tex. Meets fourth Monday of each month, except June, July, August and December, at the Doctors' Club, 8:00 p.m., Houston, Tex.

Idaho State Radiological Society

Secretary, Dr. George H. Harris, Bannock Memorial Hospital, Pocatello, Idaho. Meets in the spring and fall.

Illinois Radiological Society

Secretary, Dr. George A. Miller, Carle Hospital Clinic, Urbana, Ill. Meets in the spring and fall.

Indiana Roentgen Society, Inc.

Secretary, Dr. Richard A. Silver, 1815 N. Capitol Avenue, Indianapolis, Ind. Meets first Sunday in May and during fall meeting of Indiana State Medical Association.

Iowa Radiological Society

Secretary, Dr. L. L. Maher, 1419 Woodland Ave., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society

Secretary-Treasurer, Dr. Robert C. Lawson, 310 Medical Arts Bldg., 10th and Horne, Topeka, Kan. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter, American College of Radiology

Secretary-Treasurer, Dr. Robert H. Greenlaw, Dept. of Radiology, Univ. of Kentucky Med. Ctr., Lexington, Ky. Meets semiannually.

Kentucky Radiological Society

Secretary-Treasurer, Dr. Joan R. Hale, 402 Heyburn Building, Louisville, Ky. Meets monthly on second Friday at Sheraton Hotel, Louisville, Ky.

Kings County Radiological Society

Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 p.m.

Knoxville Radiological Society

Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society

Secretary, Dr. David Faegenburg, Nassau Academy of Medicine, 1200 Stewart Ave., Garden City, L. I., N. Y. 11533. Meets second Tuesday of the month in February April, June, October and December.

Los Angeles Radiological Society

Secretary, Dr. Joseph A. Parks, 15107 Vanowen St., Van Nuys, Calif. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif.

Louisiana-Texas Gulf Coast Radiological Society

Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Texas 77701.

Maine Radiological Society

Secretary, Dr. J. T. Chen, 7 Cherry Hill Terrace, Waterville, Me. Meets in June, September, December and April

Maryland Radiological Society

Secretary, Dr. Henry Startzman, Medical Arts Building, Baltimore, Md.

Memphis Roentgen Society

Secretary-Treasurer, Dr. Vernon I. Smith, Jr., Suite 203, 1085 Madison Ave., Memphis, Tenn. 38104. Meets first Monday of each month at John Gaston Hospital.

Miami Valley Radiological Society

Secretary, Dr. Darwood B. Hance, Reid Memorial Hospital, Richmond, Indiana. Meets third Tuesday of fall and winter months at 7:30 p.m. at Miami Valley Hospital, Dayton, Ohio.

Mid-Hudson Radiological Society

Secretary-Treasurer, Dr. Alexander W. Friedman, Mid-Hudson Medical Group, Fishkill, N. Y. Meets 7:00 p.m., first Wednesday of each month, September to May.

Milwaukee Roentgen Ray Society

Secretary-Treasurer, Dr. Donald P. Babbitt, 1700 W. Wisconsin Ave., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society

Secretary-Treasurer, Dr. Edward A. Peterson, St. Paul, Minn. Meets twice annually, fall and winter.

Mississippi Radiological Society

Secretary-Treasurer, Dr. Dan T. Keel, Jr., 504 Chippewa St., Brookhaven, Miss. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 p.m.

Missouri Radiological Society

Secretary-Treasurer, Dr. M. Shoss, Cape Girardeau, Mo.

Montana Radiological Society

Secretary, Dr. Clark Grimm, Great Falls, Montana. Meets at least once a year.

Nebraska State Radiological Society

Secretary, Dr. Richard Bunting, The Radiologic Center, Nebraska Methodist Hospital, Omaha 31, Neb. Meets third Wednesday of each month at 6 p.m. in Omaha or Lincoln.

Nevada Radiological Society

Secretary, Dr. William G. Arbonies, Department of Radiology, St. Mary's Hospital, Reno, Nev.

New England Roentgen Ray Society

Secretary, Dr. Jack R. Dreyfuss, Zero Emerson Place, Boston, Mass. 02114. Meets third Friday of each month, October through April, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 4:30 p.m.

New Hampshire Roentgen Ray Society

Secretary, Dr. Paul Y. Hasserjian, 1470 Elm St., Manchester, N. H. Meets four to six times yearly.

New Mexico Association of Radiologists
Secretary-Treasurer, Dr. Justin J. Wolfson, Department of Radiology, Bernalillo County-Indian Hospital, Albuquerque, New Mexico.

New Mexico Society of Radiologists
Secretary, Dr. William G. McPheron, Hobbs, New Mexico. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New York Roentgen Society
Secretary, Dr. Milton Elkin, Albert Einstein College of Medicine, Bronx, N.Y. 10461. Meets monthly on third Monday at the New York Academy of Medicine at 4:30 p.m. Annual meeting to be announced.

North Carolina Radiological Society
Secretary, Dr. E. H. Schultz, North Carolina Memorial Hospital, Chapel Hill, N. C. Meets in the spring and fall each year.

North Dakota Radiological Society
Secretary, Dr. Robert J. Olson, 1240 8th Ave., Williston, N. D. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. Charles H. Newell, 800 Miami Road, Jacksonville 7, Fla. Meets quarterly in March, June, September and December.

Northeastern New York Radiological Society
Secretary, Dr. Anthony J. Tabacco, 621 Central Ave., Albany 6, N. Y. Meets in Albany area on second Wednesday of October, November, March and April.

Northern California Radiological Society
Secretary-Treasurer, Dr. John Turner, 1215-28th St., Sacramento, Calif. Meets fourth Monday of Sept., Nov., Jan., March and May at the Sutter Club in Sacramento.

Northwestern Ohio Radiological Society
Secretary, Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

Ohio State Radiological Society
Secretary, Dr. Mortimer Lubert, Cleveland, Ohio. Annual meeting: Toledo, Ohio, May 13-15, 1966.

Oklahoma State Radiological Society
Secretary, Dr. Robert Sukman, 1200 N. Walker, Oklahoma City, Okla. Meets in January, May and October.

Orange County Radiological Society
Secretary, Dr. George W. Logan, 301 Newport Blvd., Newport Beach, Calif. Meets fourth Tuesday of every month at Orange County Medical Association Building.

Oregon Radiological Society
Secretary-Treasurer, Dr. Robert S. Miller, 13753 S.W. Farmington Rd., Beaverton, Ore. 97005. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary-Treasurer, Dr. Willis Taylor, 1118 9th Ave., Seattle, Washington. Annual meeting to be announced.

Pennsylvania Radiological Society
Secretary, Dr. T. Frederick Weiland, Jr., 619 Ridgeway Ave., Grove City, Pa. Annual meeting: Hotel Hershey, May 12-14, 1966.

Philadelphia Roentgen Ray Society
Secretary, Dr. C. Jules Rominger, Misericordia Hospital, 54th St. and Cedar Ave., Philadelphia, Pa. 19143. Meets first Thursday of each month at 5 p.m., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Robert N. Berk, 3305 Fifth Ave., Pittsburgh 13, Pa. Meets second Wednesday of month, October through June at Park Schenely Restaurant.

Radiological Society of Connecticut, Inc.
Secretary-Treasurer, Dr. Orlando F. Gabriele, 1450 Chapel St., New Haven 11, Conn. Meetings are held quarterly.

Radiological Society of Greater Cincinnati
Secretary, Dr. Harold N. Margolin, 6159 Tulane Road, Cincinnati, Ohio. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of Greater Kansas City
Secretary, Dr. J. Stewart Whitmore, 1010 Rialto Bldg., Kansas City, Mo. Meets last Friday of each month.

Radiological Society of Hawaii
Secretary-Treasurer, Dr. Robert W. Edland, P.O. Box 282, USA Tripler General Hospital, Honolulu, Hawaii. Meets third Monday of each month at 7:30 p.m.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana
Secretary, Dr. Lester W. Eavenson, 2700 Napoleon Ave., New Orleans 15, La. Meets semiannually, during Louisiana State Medical Society meeting and 6 months later.

Radiological Society of New Jersey
Secretary, Dr. E. Arthur Kratzman, 912 Prospect Ave., Plainfield, N. J. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of Rhode Island
Secretary-Treasurer, Dr. John M. Vesey, 1196 Elmwood Ave., Cranston, R. I.

Radiological Society of South Dakota
Secretary-Treasurer, Dr. Donald J. Peik, 303 S. Minnesota Ave., Sioux Falls, S. D.

Radiological Society of Southern California
Secretary-Treasurer, Dr. Gerald M. McDonnel, U.C.L.A. Medical Center for Health Sciences, Los Angeles, Calif. 90024. Meets three times a year, usually October, February, and May.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester 18, N. Y.

Redwood Empire Radiological Society
Secretary, Dr. Lee F. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. W. F. Hamilton, Jr., University Hospital, Augusta, Ga. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Irving B. Joffe, Rochester General Hospital, 1425 Portland Ave., Rochester 21, N. Y. Meets at 8:15 p.m. on the last Monday of each month, September through May, at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary-Treasurer, Dr. Robert W. Lackey, 4200 E. Ninth Ave., Denver, Colo. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 18-20, 1966.

San Antonio-Military Radiological Society
Secretary, Dr. Hugho F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Tex. Meets third Wednesday of each month in Fort Sam Houston Officer's Club at 6:30 p.m.

San Diego Radiological Society
President-Secretary, Charles P. Hyslop, 7901 Frost St., San Diego 22, Calif. Meets first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary, Dr. H. Joachim Burhenne, Children's Hospital and Adult Medical Center, 3700 California St., San Francisco, Calif. 94119. Meets quarterly at the San Francisco Medical Society, 250 Masonic Ave., San Francisco, Calif. 94118.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. George T. Hennessey, Washington, D. C. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 p.m.

Section on Radiology, Southern Medical Association
Secretary, Dr. Andrew F. Giesen, Jr., White-Wilson Clinic, Fort Walton Beach, Fla. Annual meeting: Washington, D. C., Nov. 14–17, 1966.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary, Dr. John L. Gwinn, Children's Hospital, 4614 Sunset Blvd., Los Angeles 27, Calif. Annual meeting: San Francisco Hilton Hotel, San Francisco, Calif., Sept. 26, 1966.

Society of Nuclear Medicine
Secretary, Mr. C. Craig Harris, Oak Ridge National Laboratories, Oak Ridge, Tenn. *Administrator*, Mr. Samuel N. Turiel, 430 N. Michigan Ave., Chicago 11, Ill. Annual meeting to be announced.

South Bay Radiological Society
Secretary, Northern Section: Dr. John H. Callaghan, 2900 Whipple Ave., Redwood City, Calif.; Southern Section: Dr. Carleton J. Wright, 2015 Clarman Way, San Jose, Calif. Meets second Wednesday of each month.

South Carolina Radiological Society
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Dakota Radiological Society
Secretary, Dr. Donald J. Peik, 1417 S. Minnesota Ave., Sioux Falls, S. Dak. Meets in spring with State Medical Society and in fall.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 4097, Mobile, Ala. Annual meeting to be announced.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society
Secretary-Treasurer, Dr. E. K. Carter, Holston Valley Community Hosp., Kingsport, Tenn. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas Radiological Society
Secretary, Dr. Herman C. Sehested, 815 Medical Arts Bldg., Fort Worth 2, Tex. Annual meeting to be announced.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital, Ann Arbor, Mich.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society
Secretary, Dr. Carlisle C. Smith, Salt Lake Genera lHospital, 2033 S. State St., Salt Lake City, Utah. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society
Secretary, Dr. John R. Williams, 160 Allen St., Rutland, Vt.

Virginia Radiological Society
Secretary, Dr. John M. Ratliff, Mary Immaculate Hospital, Newport News, Va.

Washington State Radiological Society
Secretary, Dr. Owen Marten, 930 Terry Avenue, Seattle, Wash. Meets quarterly.

West Virginia Radiological Society
Secretary, Dr. Karl J. Myers, The Myers Clinic-Broaddus Hospital, Philippi, W. Va. Meets concurrently with Annual Meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester Radiological Society
Secretary, Dr. Peter P. Brancucci, Westchester Academy of Medicine, Section on Radiology, Purchase, N. Y. Meets on third Tuesday of January and October and on two other dates.

Wisconsin Radiological Society
Secretary-Treasurer, Harold F. Ibach, 2400 W. Villard Ave., Milwaukee, Wis. Meets twice a year, May and September.

Wyoming Radiological Society
Secretary, Dr. Ronald R. Lund, 240 W. 9th St., Casper, Wyo. Meets in fall with State Medical Society and in spring on call of President.

Cuba, Mexico, Puerto Rico and Central America

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá.
Secretary-General, Dr. Roberto Calderón, Calle Central Oeste No. 218, Managua, Nicaragua, Central America. Meets annually in a rotating manner in the six countries.

Sociedad de Radiología de El Salvador
Secretary, Dr. Rafael Vaga Gómez.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1 Guatemala.

Sociedad de Radiología y Fisioterapía Cubana
Secretary, Dr. Miguel A. García Plasencia, Hospital Curie, 29 y F, Vedado, Habana, Cuba. Meets monthly at Curie Hospital.

Sociedad Costarricense de Radiologia
Secretary, Dr. James Fernández Carballo, Apartado VIII San José, Costa Rica.

Sociedad Mexicana de Radiología, A.C.
Calle del Oro No. 15, México 7, D. F.
Secretary-General, Dr. E. Alvarez Hernández.
Meets first Monday of each month.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Jorge Carreras Girard, Suite 504, Professional Bldg., Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
Secretary, Dr. R. Robillard, Notre-Dame Hospital, 1560 Sherbrooke St., East, Montreal, Que., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretary, Dr. G. H. du Boulay, 32 Welbeck St., London, W. 1, England. Meets monthly from October until May. Annual meeting to be announced.

Canadian Association of Physicists, Division of Medical and Biological Physics.
Honorary Secretary-Treasurer, Paul M. Pfalzner, Dept. of Therapeutic Radiology, University of Western Ontario London, Ont., Canada. Annual meeting to be announced.

Edmonton and District Radiological Society
Secretary, Dr. B. V. Evans, 105 Northgate Bldg., Alberta, Canada. Meets second Tuesday of each month in various Edmonton Hospitals.

Faculty of Radiologists
Honorary Secretary, Dr. J. N. Pattinson, 47 Lincoln's Inn Fields, London, W.C.2, England. Annual meeting to be announced.

Faculty of Radiologists, Royal College of Surgeons in Ireland

Registrar, Dr. H. O'Flanagan, F.R.C.P.I., D.P.H., 123 St. Stephens Green, Dublin 2, Ireland.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1, England.

Canadian Association of Radiologists
Honorary Secretary-Treasurer, Dr. D. J. Sieniewicz, *Associate Honorary Secretary-Treasurer*, Dr. Maurice Dufresne, 1555 Summerhill Ave., Montreal 25, Que., Canada. Annual meeting to be announced.

Montreal Radiological Study Club
Secretary, Dr. Leonard Rosenthall, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française de Radiologie
Secretary General, Dr. Jacques Lespérance, 1656 Sherbrooke East, Montreal, Que., Canada. Meets every third Tuesday from October to April. Annual meeting to be announced.

Toronto Radiological Society
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

College of Radiologists of Australasia
Honorary Secretary, Dr. E. A. Booth, c/o British Medical Agency, 135 Macquarie St., Sydney, N.S.W., Australia.

South America

Asociación Argentina de Radiología
Secretary, Dr. Lidio G. Mosca, Avda. Gral. Paz 151, Córdoba, Argentina. Meetings held monthly.

Ateneo de Radiologia
Secretary, Dr. Victor A. Añaños, Instituto de Radiologia, Santa Fe 3100, Rosario, Argentina. Meets monthly on second and fourth Fridays at 7:00 P.M. in the Hospital Nacional de Centenario, Santa Fe 1300, Rosario.

Colégio Brasileiro de Radiologia
Secretary-General, Dr. Tede Eston de Eston, Caixa Postal 5984, São Paulo, Brazil.

Sociedad Argentina de Radiologia
Secretary, Dr. Edwarde A. Navarrine, Santa Fe 1171, Buenos Aires. Meetings are held monthly.

Sociedad Bolivana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1596, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Nicola Caminha, Av. Mem. de Sa, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. J. P. Velasco, Avenida Santa María 0410, Santiago, Chile. Meets fourth Friday of each month.

Sociedad Colombiana de Radiologia
Secretary, Dr. Armando Uribe, Hospital Miltar Central, Apartado aéreo No. 5804, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología y Fisioterapía
Secretary, Dr. Luis Blum, P.O. Box 3712, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Miguel González Addone, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary, Dr. Vicente Ubillus, Apartado 2306, Lima, Peru. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta 218, Lima.

Sociedad de Radiologica del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiologia.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Ernesto H. Cibils, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiologia de Pernambuco
Secretary, Dr. Manoel Medeiros, Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenologia y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Carlos A. Oulton, Santa Rosa 447, Córdoba, Argentina.

Sociedad Venezolana de Radiología
Secretary-General, Dr. Luis F. Muro, Apartado No. 9362 Candelaria, Caracas, Venezuela. Meets monthly third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
President, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik. Annual meeting to be announced.

Société Belge de Radiologie
General Secretary, Prof. Simon Masy, Louvain, Belgium. Meets in February, March, May, June, September, October, November and December.

Société Européenne de Radiologie Pédiatrique
Permanent Secretary, Dr. Jaques Sauvegrain, Hôpital des Enfants-Malades, 149, rue de Sèvres, Paris 15e, France. *General Secretary*, Dr. Ole Eklöf, P.O. Box, Stockholm 60, Sweden. Annual meeting to be announced.

Société Française d'Électroradiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord. Central Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru, Paris 8^e, France.

Československá Společnost pro Roentgenologii a Radiologii
Secretary, Dr. Robert Poch, Praha 12, Srobárova 50, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Röntgengesellschaft
Secretary, Professor Dr. med. H. Lossen, Universitäts-Röntgeninstitut, Lagenbeckstr. 1, Mainz, Germany.

Società Italiana di Radiologia Medica e di Medicina Nucleare
Secretary, Dr. Ettore Conte, Ospedale Mauriziano, Torino, Italy. Meets annually.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. H. F. O. Stricker, Schalklaar, Netherlands.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association call d the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Española de Radiología y Electrología Médicas y Medicina Nuclear
Secretary, Dr. D. Aureo Gutierrez Churruca, Esparteros, No. 9, Madrid, Spain. Meets monthly in Madrid.

Schweizerische Gesellschaft für Radiologie und Nuklearmedizin (Société Suisse de Radiologie et de Médecine Nucléaire)
Secretary, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

Asia

Indian Radiological Association
Secretary, Dr. R. F. Sethna, Navsari Building, Hornby Road, Bombay 1, India.

Indonesian Radiological Society
Secretary, Professor Sjahriar Rasad, Taman Tjut Mutiah 1, Diakarta, Indonesia.

ABSTRACTS OF RADIOLOGICAL LITERATURE

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ROENTGEN DIAGNOSIS

NECK AND CHEST

MULLER, T. An unusual anomaly of the neck: a case report. *Med. Proc.*, Oct., 1965, *11*, 537–547. (From: Department of Anatomy, University of Pretoria, Pretoria, Transvaal, Union of South Africa.)

The author reports an unusual anomaly, absence of the neck, in a 7 month old Bantu fetus. This condition was in association with many other congenital anomalies, including a large diaphragmatic hernia. Roentgenograms of the head and neck region were obtained and showed a cervical vertebral column which appeared to be double rather than a bifid single column, and ribs were attached to it on the left side. In addition the right petrous bone appeared to be poorly developed.

The author relates in detail the external appearance of the fetus in addition to a detailed report of anatomic dissection of the fetus. In the head and neck area anatomic dissection showed a large anterior fontanelle approximately 5 cm. in cross section. It was asymmetric, being wider on the right side. In addition a wide gap separated the frontal and parietal bones. The parietal bones were widely separated and were unusually large. There was a distinct notch along the medial aspect of the right parietal bone. The posterior fontanelle was unusual in shape and size due to a wide separation of the two halves of the underdeveloped irregular squamous portion of the occipital bone. Here the occipital bones appeared to end abruptly on the trunk.

The major value of this paper is the discussion which is concerned with iniencephalus. The three cardinal characteristics of iniencephalus are: (1) imperfect formation of the occiput in the neighborhood of the foramen magnum; (2) spina bifida of considerable extent; and (3) retroflexion or backward bending of the spine, the face therefore looking upward.

According to Gilmour, the more extreme form of Klippel-Feil syndrome is indistinguishable from iniencephaly. The major difference between the abnormalities that range from the Klippel-Feil syndrome to iniencephalus and those included in the encephalomyelocele group, is that the brain and the cord in the former are confined in the bony skeleton and in the latter lie in a sac partially outside the bony framework of the head and spine. According to Potter, in iniencephalus the brain is often normal except for changes caused by the altered shape of the skull, although there may be dilatation of the ventricles and thinning of the cortex. In the case reported in this article the brain showed marked asymmetry, hydrocephalus and encephalocele posteriorly. The cerebellum was hypoplastic and overlay the unclosed portion of the spinal cord in the abnormal foramen magnum.

As regards etiology, this is apparently a condition initiated in a very early state (primitive streak) rather than in the more advanced embryo, causing damage to the presumptive notochord and somites of the cervical occipital region so that further ontogeny is grossly disturbed. This leads not only to inhibition, but also to a bizarre growth pattern resulting in numerous associated malformations. Severe early damage would presumably be manifested in the grossest form of iniencephalus completely incompatible with life, grading down to the milder form of Klippel-Feil syndrome.—*Lionel W. Young, M.D.*

SIMON, GEORGE. The cause and significance of some long line shadows in the chest radiograph. President's Address. *Proc. Roy. Soc. Med.*, Nov., 1965, *58*, 861–866. (From: Diagnostic Radiology Department, St. Bartholomew's Hospital, London, England.)

Kerley, Trapnell, Nelson and Fleischner *et al.*, have dealt with and explained various line shadows appearing in chest roentgenograms. The author believes that the commonest cause of a long line shadow is a part of the pleura.

He describes various appearances of the main fissure and the horizontal fissure occurring with the presence of exudate or following resection or atelectasis of a lobe. Accessory fissures may cast a line shadow but examination in posteroanterior and lateral projections should establish their true nature. The line cast by the wall of a bulla may also present as a linear or curvilinear shadow.

In cases of significant alteration of the appearance of the horizontal fissure, observation of the number and orientation of the hilar vessels may reveal an underlying atelectasis involving a lobe. The lines described as "plate atelectasis" are commonly seen postsurgically with a high diaphragm and impaired respiration. This same line may be seen in older patients without pulmonary disease. The etiology then is difficult to determine.

This is an authoritative presentation with thoughtful analysis. The Address is recommended to the reader in its entirety for more thorough explanation of the various line shadows.—*George Kling, M.D.*

MURRAY, M. J., and KRONENBERG, RICHARD. Pulmonary reactions simulating cardiac pulmonary edema caused by nitrofurantoin. *New England J. Med.*, Nov. 25, 1965, *273*, 1185–1187. (From: Department of Medicine, University of Minnesota Medical School, Minneapolis, Minn.)

Sensitivity reactions of the lung to drugs are uncommon and usually take the form of pulmonary infiltration with eosinophilia. Rarely do such reactions present a picture of pulmonary edema.

Sensitivity reaction in the lungs may occur in patients receiving nitrofurantoin and may be readily mistaken for pulmonary edema leading to incorrect management. In the 3 cases reported the physical findings did not relate well to the roentgenologic findings. There were widespread crepitant rales of the type ordinarily attributed to edema of the lungs. Two of the chest roentgenograms were clear and a third showed pulmonary congestion and a very transient consolidation of the right lower lobe. Although the white blood cell count was raised in all 3 patients, it was the neutrophils, not the eosinophils, that were responsible for the elevation. Fever was present in all to some degree, raising the possibility of pulmonary infection.

Nitrofurantoin is a bactericidal agent used in the treatment of genitourinary infections. Although a variety of toxic reactions have been reported after administration, only 5 cases of allergic pneumonitis have been recorded. All the reported cases followed the same pattern. Attacks usually began 3 or 4 days after the drug was started, and were characterized by chills, fever, cough and dyspnea. The findings consisted of cyanosis and rales. Roentgen study revealed either no abnormalities or patchy infiltrations at the lung bases. Eosinophilia was often present, but not of the order seen in eosinophilic pneumonitis. The symptoms and physical signs always exceeded the roentgenographic findings. Attacks could always be produced by rechallenging with the drug.

The patients most likely to be treated with nitrofurantoin are the elderly with chronic urinary infection. The same patients because of their age are likely to have established ischemic heart disease or to be suspected of it when the symptoms first appear. Should nitrofurantoin sensitivity be suspected, the findings of a normal circulation time would be against pulmonary edema of cardiac origin and in favor of noncardiac causes. The drug should then be stopped and the patient observed.—*David Morse, M.D.*

Holden, Helen M., Quinlan, J. J., and Hiltz, J. E. Co-existing pulmonary tuberculosis and bronchogenic carcinoma: a report of 15 cases. *Canad. M. A. J.*, Dec. 18, 1965, *93*, 1306–1310. (From: Nova Scotia Sanatorium, Kentville, Nova Scotia, Canada.)

Carcinoma of the lung as well as active tuberculosis is common in individuals over the age of 50 years. Numerous reports have been made recently on the combination of the 2 diseases in the same patient. One of 3 conclusions are usually made to explain the association: (a) tuberculosis, by its metaplastic effect on the bronchial epithelium, initiates the development of carcinoma; (b) carcinoma, during its extension in the lung, activates dormant tuberculous lesions; and (c) the occurrence of both diseases in the same patient is pure coincidence.

Since 1940, 80 patients with carcinoma of the lung have been treated at the Nova Scotia Sanatorium; in 15, coexisting active pulmonary tuberculosis was present. No characteristic clinical or roentgenologic findings indicated that the tuberculous individual also had lung cancer.

The first conclusive evidence of malignancy was provided by cytologic examination of the sputum in 6, bronchoscopic specimen in 4, pre-scalene node biopsy in 1, pulmonary resection in 1, and by autopsy in 3. Considerable time elapsed before carcinoma was suspected.

At the Nova Scotia Sanatorium, it is now the policy on all patients over the age of 50 years with tuberculosis to do repeated cytologic examinations of the sputum and to do bronchoscopic examinations.

Four patients with known cancer were considered suitable for thoracotomy. Attempt at curative resection was made in 3. One survived 7 years before accidental death; 1 is alive less than 1 year after operation, the third died as a result of the surgery.

Radiation therapy alone was given to 2 patients, 1 individual survived less than 2 months and the other for more than 1 year. One patient survived for almost 3 years following a combination of irradiation and chemotherapy.—*George A. Miller, M.D.*

Pohl, R. Der Narbenkrebs der Lunge. (Scar carcinoma of the lung.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Nov., 1965, *103*, 515–532. (Address: Lange Gasse 63, Wien VIII, Austria.)

Lung cancer arising in scar tissue is an uncommon condition and can easily be overlooked even at necropsies. It usually remains small and inconspicuous, while widespread metastatic lesions predominate. Only meticulous search for the primary tumor may lead to its detection in pulmonary scars. Nine verified cases are reported.

In the first patient, symptoms were present for 3 years. A soft tissue tumor removed from the shoulder region proved to be a metastatic carcinoma. Roentgenograms of the chest disclosed a tuberculoma of the right upper lobe. At necropsy, a carcinoma was found in this tuberculoma. Identical tumor tissue was also present in the liver and the left kidney.

The second patient was hospitalized because of intestinal bleeding. Tuberculous scars were found in both pulmonary apices. At necropsy, scar carcinoma was present bilaterally with spread to the anterior mediastinum and the fourth lumbar body.

In the third patient, a very painful polyarthritis predominated, while pulmonary symptoms were absent. A survey chest roentgenogram revealed a small fibrotic infiltrate in the right upper lobe. Eight months later, the examination was repeated because of cough and chest pain. At this time, a large pulmonary neoplasm was present in the same area with extension to the chest wall and ribs. Metastatic lesions were found in the brain and the left adrenal gland.

The fourth patient presented with fever, cough and rusty sputum. There was pulmonary scarring in the right apex with presence of a small ovoid radiolucency. A necropsy disclosed a minute neoplasm in the scar and a small cavity peripherally. Hematogenous metastatic seeding was present in the adrenals, brain, heart, stomach, thyroid gland and the peritoneal wall, but not locally.

In the fifth patient, neurologic manifestations predominated. Osteolytic lesions were present in several ribs, but the lungs were free from changes except for fibrotic strands in the right lower lobe. The anatomic diagnosis of a primary tumor in the lung met at first with difficulty because of extensive scarring and localized pleural thickening. Metastatic lesions were present in the hilar and ipsilateral lymph nodes, the brain, the adrenals and the skeletal system.

The sixth patient presented with gastric manifestations. Ulcer craters were found and were believed to be malignant, and a coin lesion in the left upper lobe was considered a metastatic process. The necropsy, however, revealed a primary carcinoma of the lung contiguous with a pleural scar. Metastatic lesions were present in the liver.

The seventh patient was operated upon because of an acute ileus. Metastatic lesions were found in the liver. A primary lung cancer was detected in tuberculous scar tissue of the right upper lobe. Metastatic seeding was also present throughout the skeletal system.

In the eighth patient, a survey roentgenogram of the chest revealed a fibrotic strand in the right upper lobe following a severe chest trauma 40 years earlier. Two years later, a large tumor was discovered in the back overlying the scar. At necropsy, widespread metastatic lesions were found throughout the body.

The ninth patient presented with (widespread) metastatic seeding throughout both lungs and many other organs. The primary tumor was found in a coin lesion of the right middle lung field.

Scar carcinoma develops primarily in old tuberculous lesions but may also be secondary to anthracosis and silicosis. Scars are subject to transformation during the course of years and decades. They are altered by mechanical, inflammatory and chemical irritants and become the sites of cholesterol deposits. Tumors in scars cannot be differentiated with certainty from peripheral bronchogenic carcinoma. Histologically, both tumor types display a wide variety.

Characteristically, the neoplasm masquerades as neuritis, neuralgia, arthritis (pulmonary osteoarthropathy), brain tumor and various other conditions. Its possibility must be kept in mind when metastatic lesions are found, and at necropsy diligent search for a minute primary lung tumor is necessary. —*Ernest Kraft, M.D.*

ABDOMEN

KLEIN, MICHAEL M., and BRADLEY, ROBERT L. (Huntington, W. Va.) Gastrospasm in gastric ulcer. *South. M. J.*, Jan., 1966, *59*, 1–9.

Gastrospasm is the most common indirect sign of gastric ulcer. Three categories of gastrospasm are observed: (1) circumscribed spasm in the form of an incisura is seen immediately opposite the ulcer, or slightly above or below, or occasionally two deep contractions are seen; (2) regional spasm is found distal to the ulcer and involves a large segment of the stomach, sometimes completely obscuring the ulcer crater; (3) total spasm diminishes the size of the stomach, with a narrowed lumen.

In the latter two types of gastrospasm, peristalsis is diminished or absent, gastric evacuation is delayed, and differentiation from an infiltrating malignancy is often difficult.

The use of antispasmodic drugs is discussed, particularly the injectable forms which act on smooth muscle contraction.—*Mark D. Reiss, M.D.*

SEMAT, P., and GRAS, F. Fistule gastro-colique par perforation d'un ulcère cortisonique de la grande courbure de l'estomac. (Gastrocolic fistula from perforation of a cortisone ulcer on the greater curvature of the stomach.) *J. de radiol., d'électrol. et de méd. nucléaire*, Nov., 1965, *46*, 741–746. (From: Service de Radiologie du Centre médico-chirurgical Foch, Paris, France.)

A cortisone ulcer typically appears at the lowest point of the greater curvature where tablets of the medication collect.

In reporting 1 new case and reviewing 12 others the authors describe this typical progression: oral cortisone therapy, appearance of fistulized ulcer without inflammatory reaction, discontinuance of cortisone, and spectacular regression of the fistula with the appearance of edema and inflammation.

In the authors' case as in some others the fistula closed spontaneously after the administration of cortisone was stopped. They note that in their case and in 5 others of the series the diagnosis could be made by study with barium meal only; in other cases barium enema examinations were required.—*Frank A. Riebel, M.D.*

EDWARDS, A. M., MICHALYSHYN, B., SHERBANIUK, R. W., and COSTOPOULOS, L. B. Regional enteritis of the duodenum: a review and report of five cases. *Canad. M. A. J.*, Dec. 18, 1965, *93*, 1283–1295. (From: Departments of Medicine, Surgery and Radiology, University of Alberta Hospital and

University of Alberta Medical School, Edmonton, Alberta, Canada.)

The frequency of regional enteritis is one-fifth that of ulcerative colitis. In 1954, review of a series of 600 cases from the Mayo Clinic revealed duodenal involvement in only 3. The literature as reported by the authors contains 43 cases of duodenal involvement and they add 5 of their own to bring the total to 48. In 2 cases of the authors' series the patient presented with symptoms of upper gastrointestinal disease. Of the 5 cases, 3 had clinical and pathologic manifestations and radiologic and operative findings of duodenal involvement early in the course of regional enteritis. The other cases showed severe involvement of the duodenum after several years of ileal disease.

In cases of duodenal obstruction, regional enteritis should be considered in the differential diagnosis. Comfort *et al.* in 1950 divided the clinical picture of patients with duodenal involvement into 4 patterns: (1) continuance of intermittent upper abdominal distress, often intensified by food, and often accompanied by loss of weight and vomiting; (2) diarrhea and steatorrhea, episodic in type; (3) gastric retention, denoted by succussion splash and by high morning gastric residue; (4) deficiency syndromes resulting from malabsorption. These patterns are not specific for duodenal disease alone and may also be present with upper jejunal partial obstruction.

The present view is that regional enteritis is a diffuse process in which the pathology varies with the stage of the disease. The prominent histopathologic features in the acute stage are lymphedema and lymphangiectasia. They may later progress to a nonspecific chronic inflammation with a granulomatous component and some fibrosis.

In cases of involvement of the duodenum and upper gastrointestinal tract, the other diseases to be considered in the differential diagnosis are peptic ulcer, lymphoma, scleroderma or amyloidosis.

Laparotomy is usually indicated for relief of the obstructive symptoms and for definitive diagnosis. The procedure of choice is a bypass operation combined with vagotomy to minimize the possibility of stomal ulceration. Steroid therapy is of value in nonobstructing upper gastrointestinal involvement, as the disease in this area "never results in perforation or fistula formation." One of the authors felt that salicylazosulfapyridine may be of as much value as prednisone in halting progression of the disease.—*George Kling, M.D.*

Wilkinson, Patricia, Pinto, Benjamin, and Senior, John R. Reversible protein-losing enteropathy with intestinal lymphangiectasia secondary to chronic constrictive pericarditis. *New England J. Med.*, Nov. 25, 1965, *273*, 1178–1181. (From: Gastrointestinal Research Unit and Medical Service, Philadelphia General Hospital, and Department of Medicine, University of Pennsylvania School of Medicine, Philadelphia, Pa.)

Since a significant portion of the daily catabolism of serum albumin occurs in the gastrointestinal tract, a considerable loss may occur if the mucosa is disordered or damaged. In cases of hypoproteinemia, if the reason is a protein-losing gastroenteropathy, a search should be made for the underlying treatable cause.

The authors state that in the past 5 years, 10 cases of hypoalbuminemia from protein-losing enteropathy due to constrictive pericarditis were reported. They describe a similar case. The enteropathy was confirmed by radioactive tracer studies and the mucosal changes were proven by peroral biopsy with the Crosby-Kugler capsule. After pericardiectomy the jejunal lymphangiectasia returned to normal. The serum albumin rose and the albumin disappearance rate became normal.

This situation was first reported by Jiminez-Diaz and associates in 1960. When Plauth *et al.* reviewed the 10 reported cases, 8 were shown to have had pericardiectomy. In 1 of these the protein loss continued. This may have been due to continued increased pressure in the right atrium.

Various theories have been postulated in these cases with some feeling that increased venous pressure may impede thoracic and intestinal lymph drainage and produce changes similar to "lymphangiectasia." The rate of lymph flow has also been shown to be important. Other authors believe that lymphatic obstruction rather than increased production is the more important factor. Lymphatic hypoplasia may also be a contributing factor.

Since more treatable cases of protein-losing enteropathy are being found, the authors feel it imperative to search out the cause when such a case is encountered. In their case, as in several others, pericardiectomy for constrictive pericarditis successfully reversed the clinical picture. They point out that medical treatment of the underlying condition has reversed the protein-losing disorder in cases of sprue, Hodgkin's disease, gastric polyp, Whipple's disease and lymphosarcoma. Dietary restriction of long-chain triglycerides has yielded improvement in protein loss due to lymphatic hypoplasia.—*George Kling, M.D.*

Ramirez, Renato F., Culp, Clyde E., Jackman, Raymond J., and Dockerty, Malcolm B. Villous tumors of the lower part of the large bowel. *J.A.M.A.*, Nov. 22, 1965, *194*, 863–867. (Address: Section of Publications, Mayo Clinic, Rochester, Minn.)

Villous tumors (villous adenomas) of the rectum have a bad reputation because they are highly prone to undergo malignant change (22–90 per cent) and they have a great tendency to recur after local removal.

The authors take issue with the approach of using these figures as an excuse for performing an abdominal-perineal resection with permanent colostomy for a benigh villous tumor.

Of 205 villous tumors, only 2 (1.0 per cent) showed superficial invasion of the muscularis propria by anaplastic cells. Their findings and those of others indicate that most recurrences are benign. A 7 per cent recurrence rate after local removal of a totally benign villous tumor does not constitute failure of therapy.

All cases were subjected to proctosigmoidoscopic examination. The observation of characteristic fronds or villi on the surface of the tumor is the primary method of diagnosis, since the fronds occasionally may not be evident histologically. The absence of 3 criteria of invasive cancer—fixation, induration and ulceration—was used in the selection of cases for conservative (local removal) treatment.

Multiple biopsies were taken and can be of diagnostic help if properly done, but superficial specimens are inadequate and the results of their examination unreliable. Biopsy specimens should be taken from the edge as well as the deep portions of the lesion. However, the authors feel that the clinical estimate is far more important than the microscopic findings.

Conservative treatment (transanal local excision, fulguration, radium application, or a combination of same) was successful in 98 per cent of 194 cases. Wide removal or destruction of surrounding hyperplastic mucosa must be done for successful therapy. Examination of surgical specimens from 11 patients treated by radical operation showed that conservative treatment probably would have been adequate, since the lesions were confined to the mucosa.—*Richard Pfister, M.D.*

Bopp, Josef. Erfahrungen mit Bilivistan in der ambulanten Praxis. (Experience with bilivistan in an ambulatory practice.) *Röntgen Blätter*, May, 1965, *18*, 199–202. (From: Röntgen-Institut der Allg. Ortskrankenkasse für den Stadtkreis Bielefeld, Germany.)

Bilivistan (Schering) was used in 150 ambulatory patients and was found to give excellent visualization of the gallbladder and bile ducts. It is a sodium and methylglucamine salt of diglycol acid-bis-(3-carboxy-2,4,6-triiodo analid) and is better tolerated than biligrafin. In 82 per cent, no reaction was experienced; 8 per cent had a slight tendency to nausea and vomiting; 10 per cent had a bitter taste, flushing, pruritus, or urticaria. No effect on the blood pressure was noted.

The usual precautions should be observed. Patients with a history of sensitivity receive an injection of calcistin (a combination of calcium and antistin). A test dose is advisable for all patients with hay fever, asthma, eczema or sensitivity to medication. Absolute contraindications include hepatorenal syndrome or poor general condition.

Each 20 cc. ampule contains 5.5 gm. of iodine in a 50 per cent water solution.

For optimum visualization of the gallbladder and bile ducts, films are exposed 90–100 minutes after the injection; additional films are exposed 30–45 minutes after a motor meal. In cholecystectomized patients, exposures are made 45 and 90 minutes after the injection.—*J. Zausner, M.D.*

George Phyllis, Young, W. B., Walker, J. G., and Sherlock, Sheila. The value of percutaneous cholangiography. *Brit. J. Surg.*, Oct., 1965, *52*, 779–783. (From: Departments of Surgery, Radiology, and Medicine, Royal Free Hospital, London, England.)

This article is the result of the authors' experience in examining 137 patients in whom percutaneous cholangiography was performed.

The indications for this type of investigation include severe jaundice of more than 1 month duration, when it is impossible to distinguish between hepatocellular disease and intrahepatic or extrahepatic obstruction of the main bile ducts. The method is also of value where jaundice, or attacks of cholangitis occur after any operation on the gallbladder, bile ducts or upper intestinal organs, or where there is a possibility of recurrent or residual stones or damage to part of the biliary tree.

The technique of the examination described is one that is used by many radiologists in this country.

The brief case histories of 7 patients and reproductions of 8 roentgenograms are included in the article.

The authors are of the opinion that percutaneous cholangiography is of value to the surgeon, even though the basic pathology is known. In this series of cases no serious complications were encountered.—*John H. Harris, M.D.*

GENITOURINARY SYSTEM

Swick Moses. The discovery of intravenous urography: historical and developmental aspects of the urographic media and their role in other diagnostic and therapeutic areas. The Fourth Ferdinand C. Valentine Memorial Lecture. *Bull. New York Acad. Med.*, Feb., 1966, *42*, 128–151. (Address: Consultant in Urology, The Mount Sinai Hospital, New York, N. Y.)

The author gives a very interesting discussion on urography and its clinical application as related to the investigation of various radiopaque media. For historic purposes he uses two separate periods in the development of these media, namely, that of the inorganic iodide radiopaque substances, administered cystoscopically, and the more recent one, that of the organically bound iodide compounds, administered intravenously, the latter being responsible for the development of excretion urography and other diagnostic fields in medicine. The author states that on this occasion he would limit himself to the present era, namely, to the development of the organically bound iodide compounds and their present role in urography.

In 1923, Rowntree and his co-workers, administering a 10 per cent solution of sodium iodide intravenously and also by mouth, reported the visualization of the urinary bladder and, in some cases, though faintly, the renal pelvis and ureter. Hryntschak, in 1928, employed a series of bromine and iodine compounds identified only by numbers, some of which were aromatic in nature. Roseno, in 1928, used a double compound-sodium-iodide-urea. These compounds, as well as all others attempted, fell into disuse because of reactions in some cases; in others, because of the extremely large dose required and the poor results.

The author gives a detailed account of his experience in 1928 under a Libman Fellowship and describes his investigations with selectan-neutral; later he helped develop the uroselectan which was followed by the development of hippuran. Hippuran, a mono-iodobenzoic acid derivative, interestingly and importantly bears similarity to our present urographic media, namely, urokon, hypaque, miokon, renografin, and conray. These are all tri-iodo-benzoic acid compounds, while hippuran, as mentioned, is a mono-iodo-benzoic acid derivative.

This is a fascinating article on the development of intravenous urography by the author who was one of the original investigators in this field.—*C. Peter Truog, M.D.*

Benness, G. T., Bullen, A., and Barker, A. Double-dose urography—renal function test. *J. Coll. Radiol. Australasia*, Oct., 1965, *9*, 234–245. (From: Department of Diagnostic Radiology, St. Vincent's Hospital, Sydney, Australia.)

The modern intravenous contrast agents for pyelography are excreted primarily by glomerular filtration and concentrated by tubular resorption of water. Rate of excretion is proportional to plasma concentration. Increasing the administered dose, therefore, results in greater concentration of the contrast material in the urine. The nephrographic effect and subsequent pyelograms are clearer. Abdominal compression may be avoided and more useful studies are obtained in patients with poor renal function.

Hypaque 50 per cent was injected in doses of 20 ml. per 70 pounds of body weight following a 12 hour period of dehydration. Comparison was made between the maximum urinary iodine concentration, plasma creatinine concentration, endogenous creatinine clearance and blood urea nitrogen. In all age groups, normal patients with good to excellent pyelograms, as judged visually, had urinary iodine concentration in excess of 54 mg./ml. and plasma creatinine concentration within normal limits.

A series of 200 consecutive cases included 89 patients who were completely normal by all criteria. Twenty-six patients had gross unilateral renal disease and the majority of these had normal function tests unless the other kidney was also abnormal. Patients with hypertension, heart disease with reduction in cardiac output, pyelonephritis and other diffuse renal disease were also evaluated. Correlation of the roentgen findings with urinary iodine concentration and other tests of renal function proved to be valuable in assessment and management of these patients.—*David Corbett, M.D.*

Vallebona, D. Tomografia ed urografia. (Tomography and urography.) *Radiol. med.*, Sept., 1965, *51*, 969–980. (From: Istituto di Radiologia dell'Università di Genova, Genova, Italy.)

After a careful review of the history of the procedure, the author considers its importance in diagnosis of lesions of the renal collecting system, parenchyma and sinus. His conclusions are based on a study of 2,000 cases.

His technique, which parallels that of Evans, is of interest. After having tested the patient for allergy, he injects a first dose of 50 per cent hypaque intravenously. Five minutes later, a conventional urogram is obtained. The second injection is made after 5 minutes, 50 cc. of 90 per cent hypaque together with 5 cc. of 20 per cent decholin being injected rapidly. The purpose of the dehydrocholic acid is to determine the circulation time. The tongue and the kidney are approximately at the same distance from the arm. When the patient is aware of the bitter taste of the decholin, in about 11 seconds, the exposures are made. The best nephrogram is obtained after 2 or 3 seconds. The remainder of the exposures is flashed at 60 to 90 second intervals.

A first consideration is that although many authors have agreed on the importance of nephrotomography, few have recognized its significance in the study of the renal sinus.

The usefulness of the combined study by urography and tomography is based on the following criteria: (1) definition of the outer surface of the kidney; (2) evaluation of the renal sinus and of the true thickness of the renal parenchyma; (3) a detailed study of the parenchyma in the nephrographic phase.

(1) The visualization of the kidney outline permits the diagnosis of: (a) generalized enlargement

(hypertrophy, hydronephrosis); (b) partial enlargement (tumors, cysts); (c) reduction in volume (renal atrophy).

(2) The detailed study of the renal sinus is of great aid in the diagnosis of diseases that modify the internal conformation of the kidney: (a) an increase of the adipose tissue (obesity); (b) atrophy of the parenchyma; (c) a disappearance of the adipose tissue because of invasion of the sinus (neoplasm).

(3) Differentiation of parenchymal tumors from cysts is possible in the nephrographic phase.—*Anthony A. Blasi, M.D.*

Leeming, W. A. A controlled study of conray 60 (methyl glucamine iothalamate) as a pyelographic agent compared against hypaque 50 (sodium diatrizoate). *J. Coll. Radiol. Australasia*, Oct., 1965, *9*, 246–251. (From: Auckland Hospital, Auckland, New Zealand.)

This study was conducted to assess the efficiency and safety of methyl glucamine iothalamate (conray 60). The drug was compared with sodium diatrizoate (hypaque 50) in a double blind study on 91 routine intravenous pyelograms.

Conray 60 is twice as viscous as hypaque 50, but both viscosity values are low and rapid injection is possible with either preparation. Conray 60 has a higher water solubility and is less likely to crystallize at low temperatures. Neither agent had a significant effect on blood pressure or pulse rate.

Side reactions were minimal and included one episode of emesis with each drug, nausea, flushing and a metallic taste. These responses were half as frequent with conray 60 as with hypaque 50. No significant difference in the diagnostic quality of the pyelograms was evident.—*David Corbett, M.D.*

Steiness, I. Hypertension due to renal artery obstruction in Eskimo girl: case of primary arteritis of aorta. *Brit. M. J.*, Nov. 27, 1965, *2*, 1291–1292. (From: Medical Department A, Rigshospitalet, Copenhagen, Denmark.)

A case report is presented of primary arteritis involving the aorta and left renal artery in a 17 year old girl.

Entering diagnosis was chronic glomerulonephritis and hypertension with a history dating back to age 11. Study seemed to exclude chronic glomerulonephritis, collagen disease, or generalized vascular disorder. Pyelography showed irregularities in the outline of the left ureter. Aortogram revealed irregularity in contour of the aorta, numerous left ureteric collateral vessels, and retrograde filling of a thin left renal artery. Split renal function tests indicated ischemia of the left kidney.

Surgical specimen of the left kidney and renal artery showed arteritis of the renal artery with occlusion. Right renal biopsy showed the arteriolar tissue to be thickened and eosinophilic.

The patient expired 1 year after surgery due to cerebral hemorrhage without relief of the hypertension, probably due to the changes in the arterioles of the right kidney secondary to the hypertension.

It is again pointed out that primary arteritis of the aorta probably is a clinicopathologic entity and that one of its manifestations is Takayashu's syndrome. There may be associated "rheumatic" disease.—*George A. Miller, M.D.*

Di Guglielmo, L., Baldrighi, V., and Marley, A. Moderni aspetti dell'angiografia renale. (Modern aspects of renal angiography.) *Radiol. med.*, Sept., 1965, *51*, 945–959. (From: Istituto di Radiologia and Istituto di Patologia Medica dell'Università di Pavia, Pavia, Italy.)

The authors present a number of cases studied by translumbar aortography, aortography by means of catheterization of the brachial artery, and selective transfemoral renal arteriography.

They offer an exhaustive review of the study of the renal circulation by other investigators.

It is concluded that aortography is a valuable method and has many indications. Renal angiography demonstrates the segmental structure of the kidney.

The direct visualization of the morphologic changes and of the pharmacologic effects is of inestimable value.

In 3 cases the authors injected 5 micrograms of adrenalin and observed an intense spasm at the level of the bifurcation of the renal artery which was modified by novocaine.

There are many disease entities in which an accurate diagnosis is feasible only through angiography. The method is also of great importance in establishing prognosis and indications for therapy.—*Anthony A. Blasi, M.D.*

Bunnell, I. L., and Greene, D. G. Rewards and hazards of selective renal arteriography. *J.A.M.A.*, Dec. 13, 1965, *194*, 1177–1180. (Address: Dr. Bunnell, 100 High Street, Buffalo, N. Y.)

Selective renal arteriography is a useful technique in the study of patients with hypertension. The percutaneous method of Ödman, with direction of a preformed catheter into each renal artery under fluoroscopic control, was employed by the authors in examination of 127 patients with high blood pressure. Selective catheterization was successful in 88 per cent of patients with failures due to pelvic arterial disease. Low pressure injections of meglumine diatrizoate disclosed 32 patients with occlusive lesions of the renal artery, 28 of which were unilateral. Twelve patients had subsequent nephrectomy or plastic reconstructive renal artery surgery. Seven were cured and 3 showed significant improvement. Only 1 of the 20 patients treated medically has shown any improvement.

The complication rate was quite low compared to other series with no mortality and 6 per cent morbidity. The complications included 1 dissection of the wall of the common femoral artery, 2 femoral hematomas and 3 small peripheral emboli. No permanent disability resulted in any instance. Renal function studies, including glomerular filtration rate and renal plasma flow, were unaltered by the procedure.

Diagnostic clarity is excellent, particularly since the confusing vascular shadows of the standard aortogram are absent. Greater detail of the fine renal vasculature is achieved, so that disease affecting minor branches may be appreciated.—*David Corbett, M.D.*

Figueroa, Julio E., Rodríguez-Antúnez, Antonio, Nakamoto, Satoru, and Kolff, Willem J. The scintigram after renal transplantation in man. *New England J. Med.*, Dec. 23, 1965, *273*, 1406–1410. (From: Department of Artificial Organs and Department of Radiation Therapy and Isotopes, Cleveland Clinic Foundation, Cleveland, Ohio.)

One of the principal uses of radiomercury-labeled chlormerodrin scanning of the kidneys is to show renal tubular function in pictorial form.

The authors have employed this scan test to evaluate the function and viability of 32 renal allografts, 27 of which were obtained from cadaver donors.

As might be expected, the method is helpful in evaluating the prognosis for the transplanted kidney. Failure of any of the tracer to localize within the renal substance suggests interference with perfusion due to vascular thrombosis. Impairment of tubular function in all, or a part, of the graft will be visualized as defects due to diminished counting rates in the affected areas.

The test is a useful one to add to those currently employed to study the fate of transplanted kidneys. —*Frederick J. Bonte, M.D.*

Schiavetti, E. La diagnosi radiologica del tumore di Wilms. (The radiologic diagnosis of Wilms' tumor.) *Ann. radiol. diag.*, 1965, *39*, 107–164. (From: Ospedale dei Bambini di Milano, Milano, Italy.)

The author offers a study of 23 cases of Wilms' tumor, of which 18 were confirmed operatively; the others, although not treated surgically, presented a typical clinical and radiologic picture.

The diagnosis of Wilms' tumor for the most part is not beset with difficulties. With relatively simple investigative procedures such as urography and pneumoretroperitoneum one may obtain sufficient data as to the site of origin, the progression and the nature of the abnormal tumefaction.

Urography is to be considered the principal diagnostic means. There are early cases in which this method is inadequate.

There are two main causes that may compromise the diagnosis in limited tumoral forms. One is due to the difficulty of obtaining well-detailed roentgenograms in infants. The other is that in some instances, although the tumor is of fair size, it does not produce appreciable changes in the renal pelvis.

The author believes that retrograde pyelography should only be performed if necessary for diagnostic or therapeutic considerations.

The differential diagnosis between Wilms' tumor and renal cyst may be difficult to establish. Aortography, permitting a study of the renal vascularization will give some information. One must consider that this method is not devoid of risks and must be employed judiciously.

An important problem is the differentiation of renal and extrarenal retroperitoneal tumors. Rarely, sympathicoblastomas may invade the renal parenchyma and make the correct diagnosis unattainable. —*Anthony A. Blasi, M.D.*

Nervous System

Andersson, Hugo, and Sullivan, Lars. Diastematomyelia: report of two cases submitted to laminectomy. *Acta orthop. scandinav.*, 1965, *36*, 257–264. (From: Neurosurgical Clinic, University of Gothenburg, Sweden.)

The authors report 2 cases of diastematomyelia and give a short review of the main features and treatment of the condition which is a rare congenital anomaly associated with spina bifida.

Diastematomyelia represents a bifid state of the spinal cord. The cord may be split by bony or fibrous tissue that extends from the vertebral body toward the lamina in a sagittal direction or a similarly localized area may be divided longitudinally without associated interposed tissue. In the former situation the two parts of the cord have a dual sheath of their own. In the latter situation aberrant tissue or even neoplasm may rarely interpose.

A distinction is made between diplomyelia and diastematomyelia. The former means complete doubling of the spinal cord in a localized sector, whereas the latter is splitting or division of the spinal cord. Many authors, however, do use the terms synonymously.

Although the majority of cases with diastematomyelia manifest no clinical signs, some cases do present with neurologic deficits in the lower limbs, bladder and bowel apparently secondary to prevention of the normal ascent of the spinal cord within the vertebral column as well as lateral pressure from the splitting structure. It is pointed out that the clinical signs are nonspecific for diastematomyelia since they may also occur in different types of spinal dysraphism, either alone or combined with diastematomyelia. The earliest and most frequent manifestation is usually retarded unilateral growth of one leg or foot often combined with a varus deformity. Sensory loss, trophic ulceration and functional disability of the bowel and bladder may also occur.

A radiologic investigation of the spine is indicated when there are clinical signs of spinal dysraphism. Plain roentgenograms in cases with diastematomyelia demonstrate widening of the spinal canal corresponding to the cleft bulbous widened cord and the extent of the spina bifida and sometimes additional anomalies like hemi- and fused vertebrae. The diagnosis is definite if a sagittally directed bone septum is present. In cases with no interposition or with a fibrous septum not sufficiently calcified to show on plain roentgenograms, myelography is needed for the diagnosis. In addition, this modality gives information as to the site and extent of the anomaly. Gas myelography is preferred to positive contrast myelography. This is because it provides the most accurate anatomic delineation of the entire anomaly and it does not include the risk of arachnoiditis. The gas is quickly and totally absorbed.

The authors point out that cases with diastematomyelia and neurologic signs of spinal dysraphism require surgical correction of the anomaly. An early intervention is recommended since the neurologic deficits are progressive and there is increasing risk of irreversible damage to the cord. There has been some degree of improvement in approximately two-thirds of patients operated.—*Lionel W. Young, M.D.*

SKELETAL SYSTEM

Wissinger, H. Andrew, McClain, Edward J., and Boyes, Joseph H. Turret exostosis: ossifying hematoma of the phalanges. *J. Bone & Joint Surg.*, Jan., 1966, *48-A*, 105–110. (Address: Dr. Wissinger, 128 North Craig Street, Pittsburgh, Pa.)

Following relatively minor lacerations to the dorsum of the fingers, a bony mass may develop at the site of injury, which is hard, immobile, occasionally tender, and always severely limits flexion of the involved finger. Serial roentgenograms reveal initial soft tissue injury with progressive periosteal reaction, which, 4 to 6 months following injury, can be seen as a clearly delineated bony exostosis.

Pathogenesis is believed to be an initial small periosteal defect with development of a subperiosteal hematoma, which cannot drain owing to the small size of the initial wound. As the intact periosteum overlying the hematoma is only minimally injured, and its osteogenic properties remain, gradual ossification of the hematoma occurs.

The corrective surgical procedure is described. It is stressed that excision should not be performed prior to 4 to 6 months following injury, when the exostosis is mature, and has distinct margins, without fragmentation. Premature excision invites recurrence. Of 8 patients with adequate follow-up, 5 had full return of motion and 3 were improved. All patients had relief of pain.—*Mark D. Reiss, M.D.*

Fock, G., and Sulamaa, M. Congenital short femur. *Acta orthop. scandinav.*, 1965, *36*, 294–300. (From: Children's Clinic, University Central Hospital, Helsinki, Finland.)

The authors report 12 cases of congenital unilateral hypoplasia of the femur. The cases are divided into 3 main groups on the basis of the degree of involvement and shape of the femur. There were 6 cases which showed only hypoplasia with the shape of the femur essentially normal. There were 3 cases of hypoplasia in association with coxa vara deformity and there were 3 cases with a partial absence of the upper end of the femur or a "pseudoarthrosis" in that area.

The aim of the study of these cases was to investigate the growth capacity of a congenitally short femur and to evaluate the effect of treatment on growth. In some of the cases other typical "thalidomide" deformities (*e.g.*, aplasia of the ulna on one side) were also present.

The authors measured shortening of the femur on the roentgenogram as a percentage of the length of the healthy femur. In the hypoplasia group at the beginning of treatment the shortening of the femur varied from 80 to 94 per cent. In the varus-shaped femurs the range was 62 to 77 per cent. In the group with a deficit of the upper end of the femur the percentage shortening was even greater except in 1 case. The follow-up period, counted from the initiation of treatment, ranged from 12 to 74 months. In all cases growth led to an increase in the absolute difference in length as compared with the contralateral femur, even though the relative difference either remained as before or changed for the better.

Method of treatment involved osteotomy for the varus deformity and one case showed improvement with transplantation of bone and epiphyseal cartilage to the upper femur. Detachment of periosteum and introduction of steel screws was used in the majority of cases but this was not shown to cause any significant stimulation of growth. When the length difference was not marked, the method of correction resorted to was an orthopedic shoe.

In the authors' opinion there is no indication to perform amputation in early childhood in these cases.—*Lionel W. Young, M.D.*

Cole, Arthur R. C. Mastocytosis: report of a case in a 15½-week-old infant with cutaneous and generalized skeletal changes. *Arch. Dis. Childhood*, Dec., 1965, *40*, 677–683. (From: The Princess Margaret Hospital, and Hospital for Sick Children, Toronto, Ontario, Canada.)

A case of a 4 month old infant with cutaneous and generalized skeletal mastocytosis is reported.

Patients with mastocytosis present with cutaneous, systemic or skeletal changes. The disease is encountered most frequently in infants and young children. The prognosis is good and a spontaneous recovery usually occurs between 6 to 10 years of age.

The cutaneous rash consists of red, pink, brown, and melanotic discolorations.

Systemically, spontaneous histamine shock and gastroenteric symptoms occur.

Generalized or local roentgenographic skeletal changes are common. Generalized cystic osteoporosis of the ribs with thickening of the bone trabeculae, stippling of the bone structures of the skull, thickening of the skull tables and osteosclerosis of the pelvic bones and vertebrae comprise the generalized roentgenographic changes. Locally, calcified and decalcified areas are seen in the humerus, radius, femur, skull, clavicle and scapula.—*John H. Harris, M.D.*

Zampa, Gian A., and Zucchelli, Pietro C. Pseudohypoparathyroidism and bone demineralization: case report and metabolic studies. *J. Clin. Endocrinol. & Metabol.*, Dec., 1965, *25*, 1616–1624. (From: Endocrine Clinic, Institute of Medical Pathology, University of Bologna, S. Orsola Hospital, Bologna, Italy.)

An 8 year old white boy with the full-blown picture of pseudohypoparathyroidism associated with radiologic evidence of bone demineralization and increased serum alkaline phosphatase is reported.

The unusual finding of bone demineralization has been described in about 25 per cent of confirmed cases of pseudohypoparathyroidism.

Metabolic studies were performed and their significance is discussed.

The authors postulate that an excess of parathyroid hormone is present in these cases and, in addition, a defect in the renal tubular response to parathyroid hormone may be assumed.—*John H. Harris, M.D.*

Béraud, Cl., Picard, Y., and Picard, D. Granulome éosinophile vertébral chez l'enfant (évolution radiologique). (Vertebral eosinophilic granuloma in the child [radiologic evolution].) *J. de radiol., d'électrol. et de méd. nucléaire*, Oct., 1965, *46*, 637–640. (From: Service d'Electroradiologie de l'Hôpital Debrousse, Chemin Saint-Irénée, Lyon, France.)

A number of interesting elements were met in a case of vertebral eosinophilic granuloma: onset with abdominal and vertebral pain in the absence, initially, of any roentgenologic findings; the appearance of paraplegia with sphincteral disorders; development of a lacunar lesion of the 12th dorsal vertebra with a small paravertebral spindle; the secondary discovery of a costal lesion; and finally the dramatic influence of roentgen therapy (50 r×12).

The spinal lesion became stabilized in the pattern known as Calvé's vertebra plana.—*Frank A. Riebel, M.D.*

Piggott, Harry. Radiological prediction of avascular necrosis in femoral neck fractures. *Brit. J. Surg.*, Sept., 1965, *52*, 675–678. (From: Royal Orthopaedic Hospital, Birmingham, England.)

Avascular necrosis of the femoral head can be a significant complication of femoral neck fractures. Consideration of the anatomy of the intracapsular part of the femur and its vessels suggests that it should be possible to know, from certain roentgenographic features of the fracture, when this extreme degree of vascular damage has occurred.

The vessels concerned are the lateral and medial epiphysial and the inferior metaphysial. Survival of the important superolateral weight-bearing area depends principally on the integrity of the lateral epiphysial vessels. Although the portions of the vessels extending along the posterosuperior aspect of the femoral neck are quite mobile, this mobility ceases as the vessels enter the bone through the vascular foramina. Should a fracture extend through the bone and foramina, injury of the vessels in this location seems inevitable. Thus, if the relationship of the fracture to the lateral epiphysial foramina can be determined, it should be possible to select cases in which the incidence of avascular necrosis will approach 100 per cent. The foramina cannot be seen roentgenographically but one landmark can easily and invariably be identified on a lateral roentgenogram after reduction of the fracture. This is the junction of the head and neck posteriorly, where the limit of the articular surface is marked by an abrupt change in outline from convex to straight or concave. Inspection of the upper aspect of the femoral neck will show that a line drawn transversely across the bones from this point passes through the area occupied by the lateral epiphysial foramina.

It is important to realize that the relationship of the fracture to the transverse subcapital line and thus to the foramina can be seen on only good quality roentgenograms made after reduction.

Avascular necrosis seems to be probable in fractures in which: (1) the initial anteroposterior roentgenogram shows external rotation and upward displacement sufficient to indicate that cortical continuity is completely disrupted; and (2) the postreduction lateral roentgenogram shows the fracture on, or proximal to, the transverse subcapital line.

In a series of cases studied the incidence of avascular necrosis in displaced transforaminal fractures was 88 per cent and Smith-Peterson nailing was foredoomed to failure. In a series of 57 subcapital fractures those exhibiting a fracture through the transforaminal region accounted for 80 per cent of all cases of avascular necrosis.

Preoperative reduction and a good quality lateral roentgenogram to localize the site of the subcapital fracture as described above may be of value in predicting cases where avascular necrosis is inevitable. Appropriate modification of surgery with substitution of the femoral neck with an appropriate prosthesis would then be indicated.—*David Morse, M.D.*

Kučera, J., Lenz, W., and Maier, W. Malformations of the lower limbs and the caudal part of the spinal column in children of diabetic mothers. *German Med. Monthly*, Oct., 1965, *10*, 393–396. (From: Institute for the Mother and Child, Prague, Czechoslovakia, the Institute of Human Genetics, University of Hamburg and the Surgical and Orthopaedic Department of the Paediatric Clinic, University of Munich, Germany.)

The relationship between maternal diabetes and congenital malformations has not been fully explained. However, among children of diabetic mothers a group of malformations occurs with particular frequency.

These malformations constitute a syndrome which is characterized by the absence or underdevelopment of the caudal vertebral column and femora. This may be combined with malformations of various organs as well as with polydactyly and syndactyly of the toes.

At present an exact definition of the syndrome is impossible and the precise pathogenic agent is uncertain. The correlation of the syndrome of caudal regression with diabetes does not imply that it is caused by the diabetes or its treatment. The actual deformities may be produced by lesions affecting the developing blood vessels. Such lesions are common in diabetes but also occur in other conditions.—*David Morse, M.D.*

Linscheid, Ronald L., and Wheeler, Donald K. Elbow dislocations. *J.A.M.A.*, Dec. 13, 1965, *194*, 1171–1176. (Address: Section of Publications, Mayo Clinic, Rochester, Minn.)

A statistical analysis is presented of 110 elbow dislocations where the dislocation was considered the primary injury. Sixty-four per cent of the cases received their entire care at the Mayo Clinic. Cases were excluded where significant interruption of the ulnar articular surface occurred. Two anterior and 108 posterior dislocations were seen.

Rupture of the collateral ligaments commonly occurred with dislocation associated with late heterotopic bone formation in the ligaments. Common accessory fractures involved the epicondyles and radial head. Fourteen patients received associated injuries, of which 9 occurred at the wrist. The ulnar nerve was injured approximately 3 times more commonly than the median nerve. Arterial injuries were found in 6 dislocations. All arterial injuries were associated with persistent neurologic findings. Poorer results in treatment were found in those patients with more serious initial injuries and complications. Ninety-eight patients had sufficient follow-up data; the results were considered excellent in 40, good in 29, fair in 22, and poor in 7.—*George A. Miller, M.D.*

Kernwein, Graham A. Roentgenographic diagnosis of shoulder dysfunction. *J.A.M.A.*, Dec. 6, 1965, *194*, 1081–1085. (Address: Suite 601, Gas and Electric Building, Rockford, Ill.)

The author compared conventional roentgenograms with 200 arthrographic studies of the glenohumeral joint in order to establish a connection between certain commonly seen degenerative changes and pathologic changes in the rotator cuff.

Cystic changes within the tuberosities of the humerus are discussed first. It is stated that these result from disuse atrophy of bone trabeculae in the presence of a torn rotator cuff. Cystic changes were found in 26.6 per cent of shoulders with arthrographic evidence of an incomplete tear of the rotator cuff, and in 54.6 per cent of those exhibiting a complete tear. The author believes that when cystic changes are seen in the tuberosities, there is over a 70 per cent probability that an associated attritional change exists within the rotator cuff.

Another degenerative change that was studied was erosion of the tuberosities of the humerus. This was found to be due to friction between the tuberosities and the undersurface of the coraco-acromial arch during abduction and external rotation. In time, changes in the acromion occur, such as hypertrophy and peripheral spur formation. This can progress to complete disappearance of the rotator cuff and associated soft tissue structures.

Degenerative changes in the acromioclavicular joint also appear to be due to rupture or evulsion of the rotator cuff. In this study, 85 per cent of patients with degenerative changes of the acromioclavicular joint presented with arthrographic evidence of either an incomplete or complete tear of rotator cuff.

It was also found that roughening and overgrowth of bone on the inferior surface of the glenoid fossa was due either to degenerative change or attritional change within the rotator cuff. In 92 per cent of those patients with degenerative changes of the humeral head, a lesion of the rotator cuff was demonstrated on the arthrogram.

Asymptomatic calcifications of the rotator cuff occur in areas of degeneration. These calcific masses can rupture spontaneously and produce incomplete or complete tears of the rotator cuff. This appears to be an important precursor of attritional rotator cuff lesions.

The author concludes that any four of these degenerative changes indicate a 90 per cent probability that arthrographic study will reveal a ruptured rotator cuff.—*Kenneth M. Nowicki, M.D.*

Robinson, Richard G. Massive protrusions of lumbar disks. *Brit. J. Surg.*, Nov., 1965, *52*, 858–865. (From: Neurosurgical Unit, Dunedin Hospital, and Department of Surgery, University of Otago, Dunedin, New Zealand.)

Five hundred surgically verified lumbar disk lesions were analyzed as to sex, age, and type of lesion. Fifty-two of these cases demonstrated complete or subtotal block of the subarachnoid space on myelography. Of these 52 cases, 17 demonstrated cauda equina compression syndrome. Subtotal block was considered when the subarachnoid space was reduced in volume to that of the contained roots of the cauda equina. The total series demonstrated that more males than females are affected, and the peak age incidence was between 31 and 40. In the 52 patients with massive disk protrusions, the incidence of male patients was even higher, and the age group tended to be older. In massive disk protrusions, the most common disk space was the 4th lumbar. Of the 3rd lumbar disk herniations, nearly a third were massive in type. Myelographically, the 4th and 5th lumbar levels demonstrated total and subtotal blocks in equal frequency. However, all of the blocks at the 3rd lumbar disk space were complete. In the group of patients with cauda equina compression, all but 1 had complete blocks on myelographic examination.

Clinically, the cerebrospinal fluid protein was the only differentiating finding between a total block case and a subtotal one before myelography was performed. In this group, all the symptoms were unilateral. In most of the cases of cauda equina compression previous attacks of backache and sciatica were present. The cauda equina syndrome itself usually came on within a few hours, and rarely, more than 2 days. The importance of myelography in evaluating cauda equina syndrome is stressed due to close similarity clinically between disk syndromes and transverse myelitis.—*John Hodak, M.D.*

Jones, John Paul, Jr., and Sakovich, Leo. Fat embolism of bone: a roentgenographic and histological investigation, with use of intra-arterial lipiodol, in rabbits. *J. Bone & Joint Surg.*, Jan., 1966, *48-A*, 149–164. (From: Department of Orthopaedic Surgery and the Rheumatic Disease Group, Department of Medicine, University of California School of Medicine, San Francisco Calif.)

A catheter was passed retrograde into the aorta of 26 rabbits and an average of 1.3 ml. of lipiodol infused. The right femur and tibia were examined roentgenographically and histologically at intervals varying from 0 minutes to 10 weeks following infusion.

Lipiodol emboli were demonstrated histologically up to 5 weeks, although essentially no emboli were seen roentgenographically after 7½ days. For the first 24 hours most of the lipiodol emboli were predominantly confined to metaphyseal and marrow vessels. Within the next 10 days the number and size of intravascular emboli decreased in the metaphyseal regions and increased in the subchondral bone.

The authors speculate on the possible role of systemic fat embolism in the production of avascular necrosis of bone.—*Mark D. Reiss, M.D.*

Blood and Lymph System

Ruggiero, Giovanni. Évolution de la technique de l'angiographie cérébrale. (The evolution of cerebral angiographic technique.) *J. de radiol., d'électrol. et de méd. nucléaire*, Oct., 1965, *46*, 595–606. (From: Service de Neuroradiologie, Hôpital Maggiore, Bologna, Italy.)

The cerebral arteries should be injected with selectivity as befits the case, reports the author.

Thus the common carotid artery is injected when there is presumptively an extracranial vascular accident.

There are rare but indispensable requirements when the external carotid artery only is to be filled; for instance, meningiomas of the tentorium or falx are sometimes demonstrable in this way only, or the exact site of origin of other meningiomas may be best determined by this approach.

Extradural and subdural hemorrhages may be differentiated according to the success of visualizing individual meningeal arteries; in arteriovenous fistulas prognosis is affected by the degree the external branches contribute to the circulation.

The internal (rather than the common) carotid artery should be injected when the brain supplied by it is to be studied, both to avoid confusion from external branches and because there is a higher probability of filling the posterior cerebral artery.

In vertebral angiography it is frequently advisable to do bilateral studies, for the posterior inferior cerebellar artery is not usually filled by a contralateral injection, as it originates from the vertebral artery. Incidentally, in vertebral angiographic studies a strict anteroposterior view is preferable to a semiaxial one.

In biplane studies the author prefers simultaneous exposures to the slightly alternated ones, which some workers favor to avoid secondary radiation. Preliminary roentgenograms are made in the interest of precision, and subtraction techniques have been employed in 250 cases.

Repeated angiography is favored whenever there is need, since pathology frequently is uncovered at a later date, after cerebrovascular spasm has been relieved.

In each angiography case an accurate operative record is kept, listing the surgeon, assistants, technicians, the arteries injected and the type and amount of contrast material used, with chronologic description of the progress and reactions.—*Frank A. Riebel, M.D.*

ELKE, M., and LUDIN, H. Drainage der unteren Körperhälfte bei Agenesie und erworbenem Verschluss der Vena cava caudalis. (Drainage in the lower half of the body in agenesis and acquired occlusion of the inferior vena cava.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Dec., 1965, *103*, 665–674. (Address: Univ.-Institut für Röntgendiagnostik und Strahlentherapie, Bürgerspital, Basel/Switzerland.)

Congenital anomalies of the inferior vena cava are less common than those of the superior vena cava. The former are associated with various cardiac defects in 0.3–0.6 per cent of the cases. The crucial developmental period is during the 3rd–8th week of fetal life. With closure of the superior and inferior vena cava or occlusion of the azygos system, deep and superficial venous collaterals are developed to merge with the portal system or unite with mesenteric and thoracic or subclavian venous channels. After acquired occlusion of the inferior vena cava, drainage of the lower half of the body occurs through dilatation of 3 venous channels: azygos, portal and subclavian systems.

Five patients had angiographic studies with the following findings:

(1) A 6 year old girl had a poorly developed inferior vena cava at the infrarenal, renal and hepatic segments. The renal veins emptied into a persistent supracardinal stem; the hepatic veins emptied directly into the right atrium; an atrioventricular septal defect with a left-to-right shunt was present. Venous drainage from the lower half of the body occurred through a dilated azygos system.

(2) A 2 year old girl had situs inversus visceralis totalis, aplasia of the inferior vena cava, a persistent left superior vena cava and a truncus arteriosus or variant of a tetralogy of Fallot. Venous blood from the lower half of the body emptied into a right and left superior vena cava, which emptied into the right atrium. Hepatic veins were filled in retrograde fashion from the right atrium. Pulmonary arteries were not present so that the lungs were supplied by bronchial arteries arising from the common trunk.

(3) A 56 year old woman had a postpartum thrombophlebitis 24 years prior to this study; numerous varicose veins were distributed over the lower extremities and abdomen. Angiography showed very small iliac veins and stenosis of the inferior vena cava; dilatation of a presacral hemorrhoidal plexus; dilated pre- and paralumbar veins united with the azygos system and the superior vena cava; dilated inferior mesenteric and tortuous ileocolic vessels emptied into the portal system, hepatic veins and thence into the right atrium; extensive collaterals of the abdominal and thoracic wall emptied into the azygos and subclavian veins.

(4) A 27 year old male had had a nephrectomy following a serious abdominal wound; the inferior vena cava was sutured. Angiographic studies were similar to Case 3.

(5) A 47 year old man had nephrectomy for carcinoma. The venous flow was similar to the previous patient.

The authors estimate that it takes about 1–3 weeks for a collateral circulation to be established for adequate drainage of the lower half of the body in cases of acquired occlusion of the inferior vena cava. The caliber of the venous channels varies directly with the amount of back pressure on the individual systems.—*J. Zausner, M.D.*

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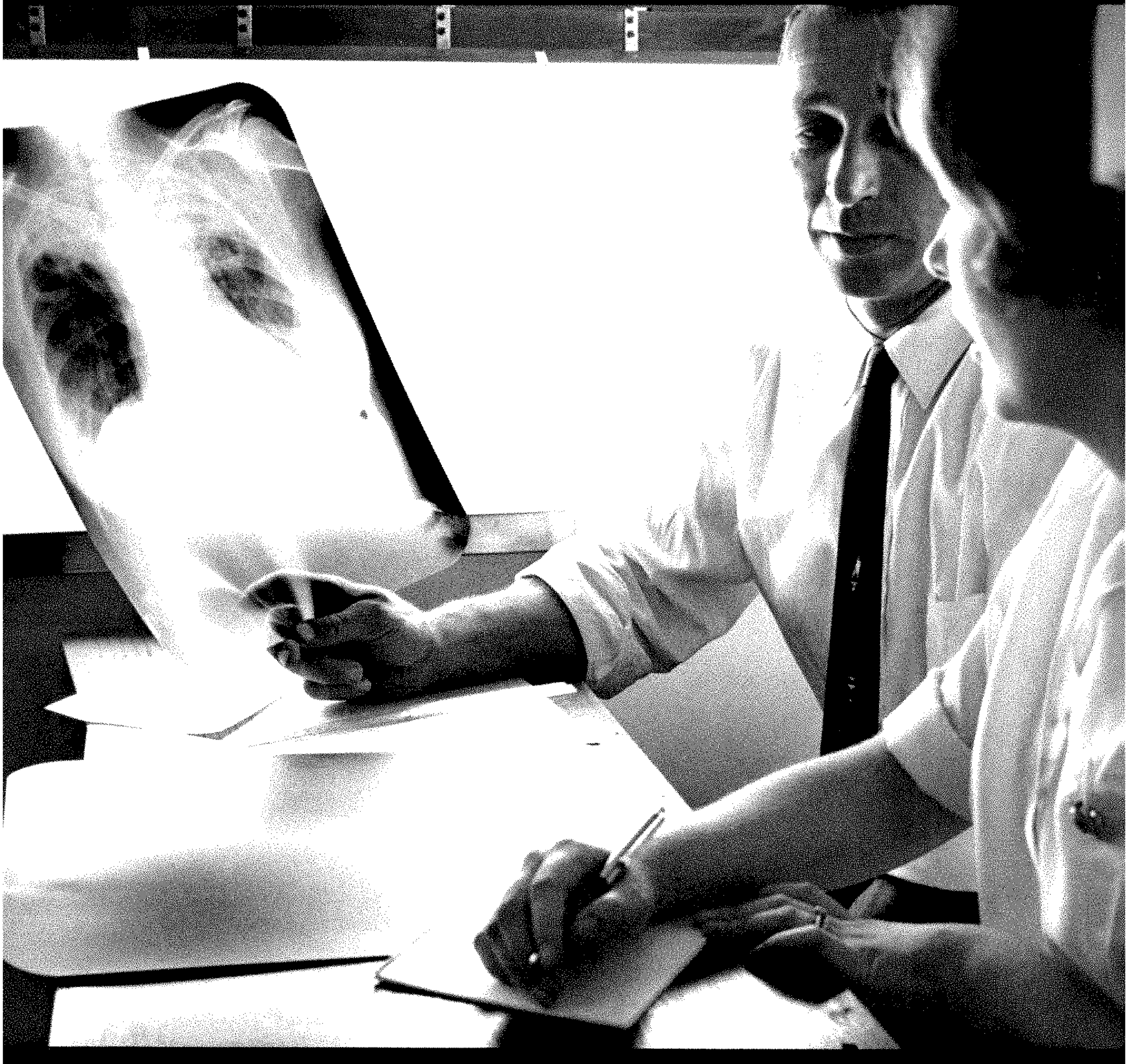

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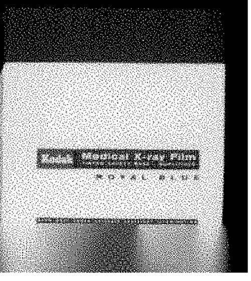

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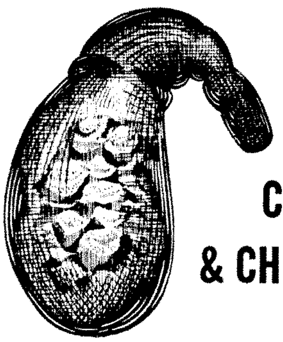

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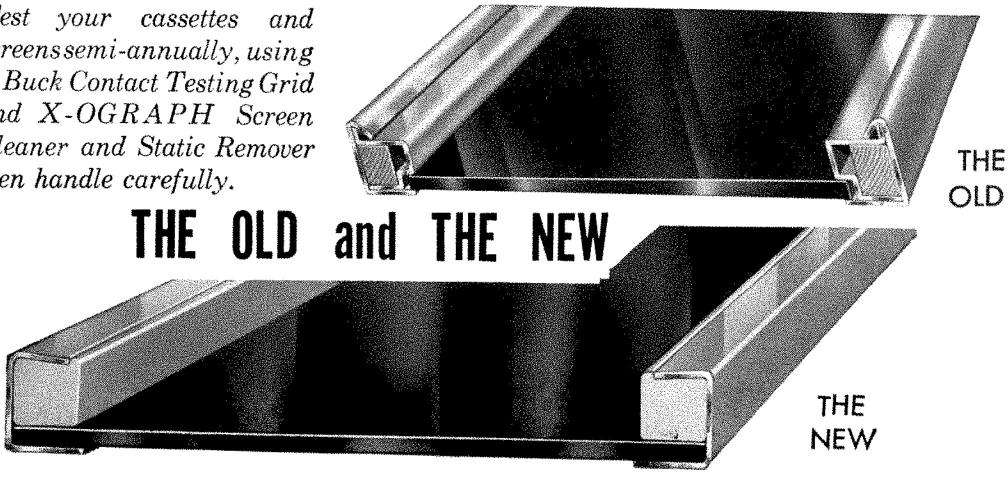

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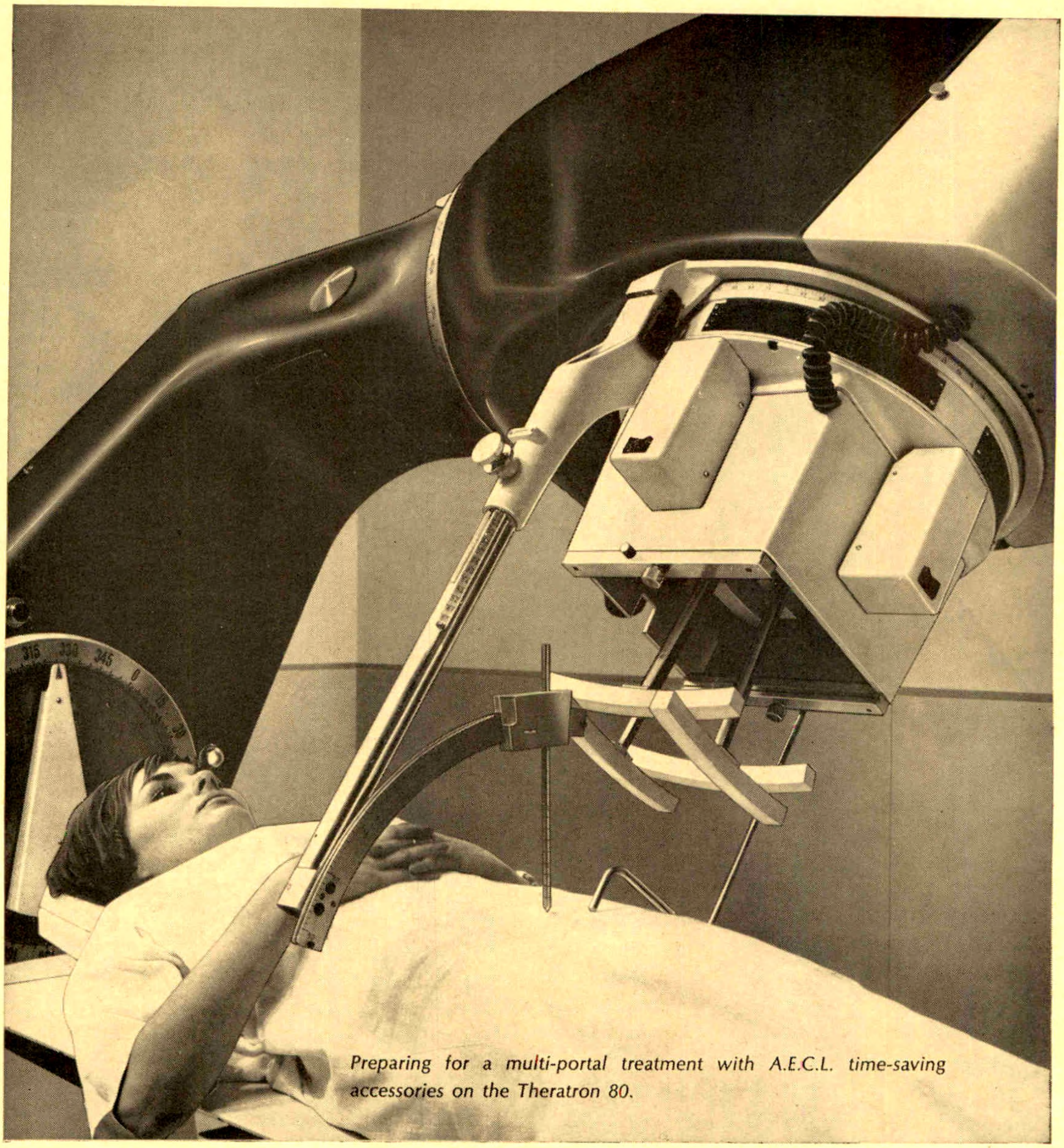

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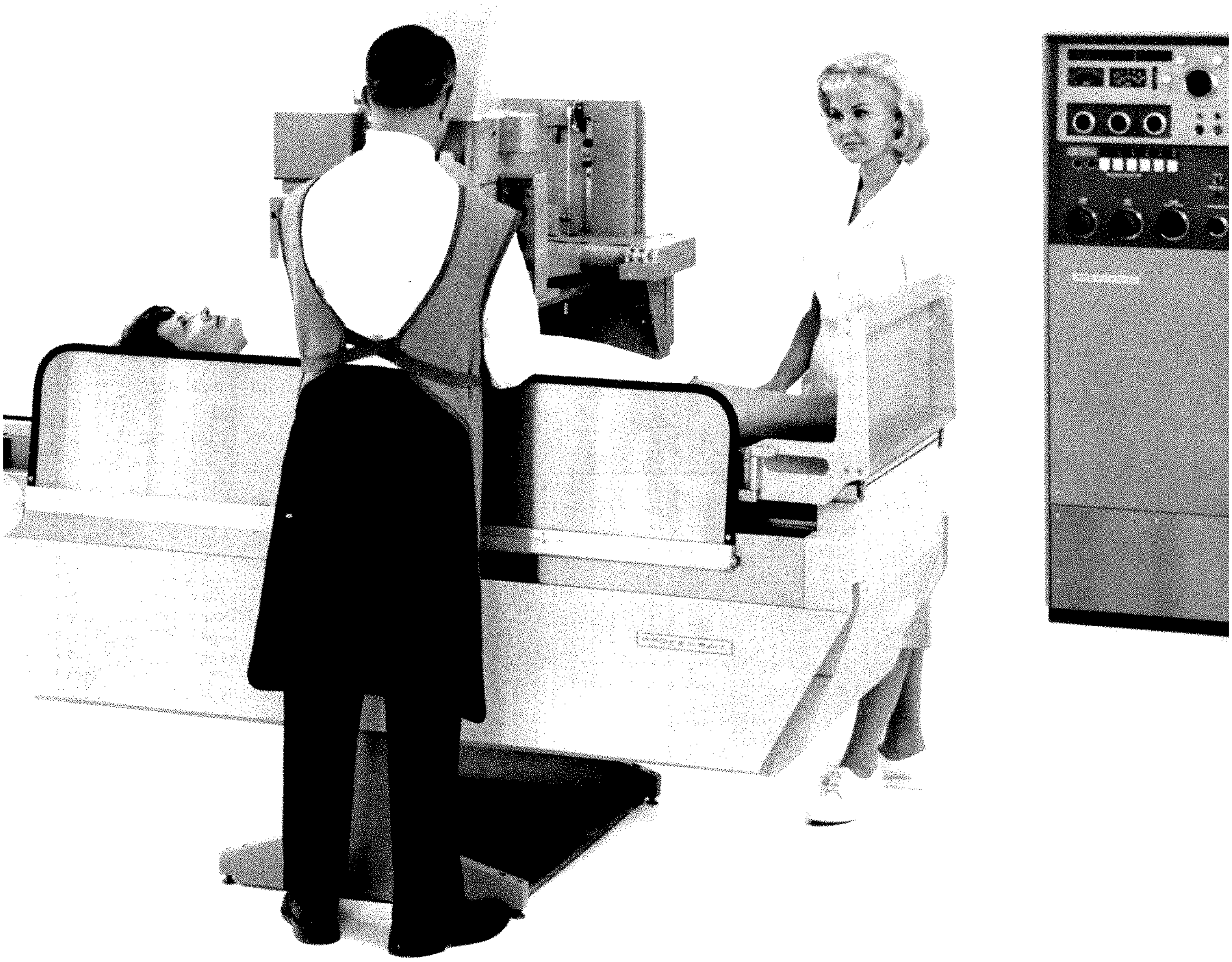

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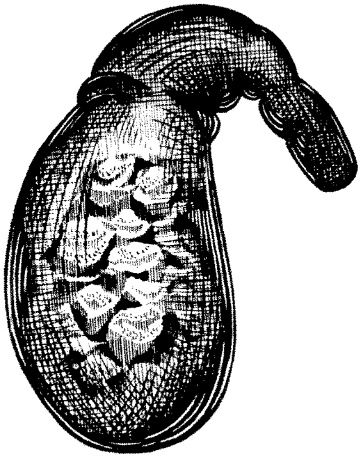

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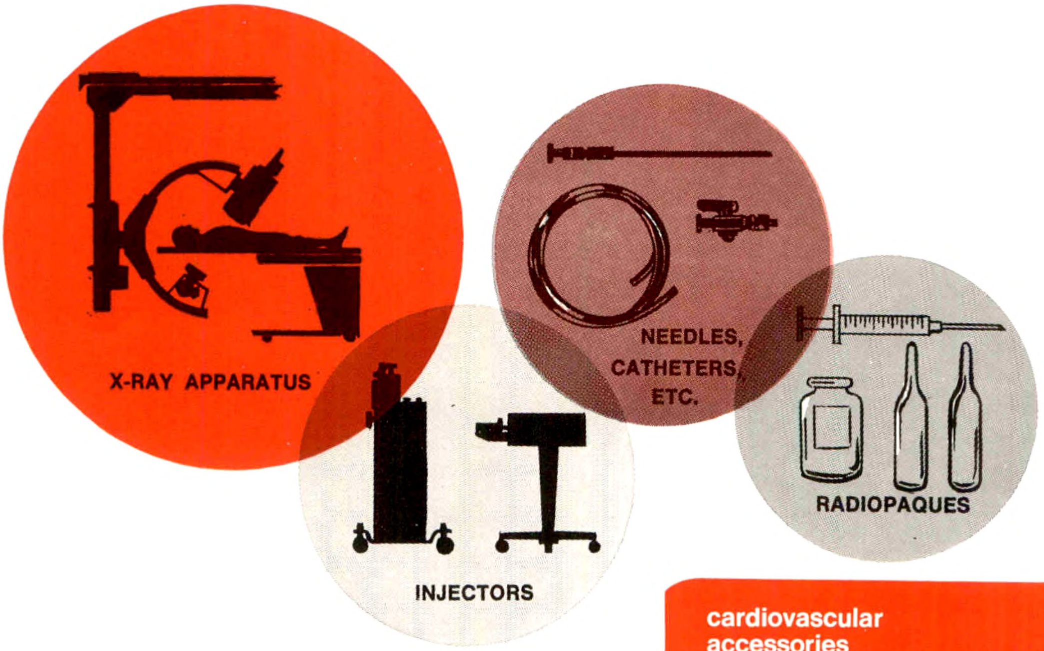

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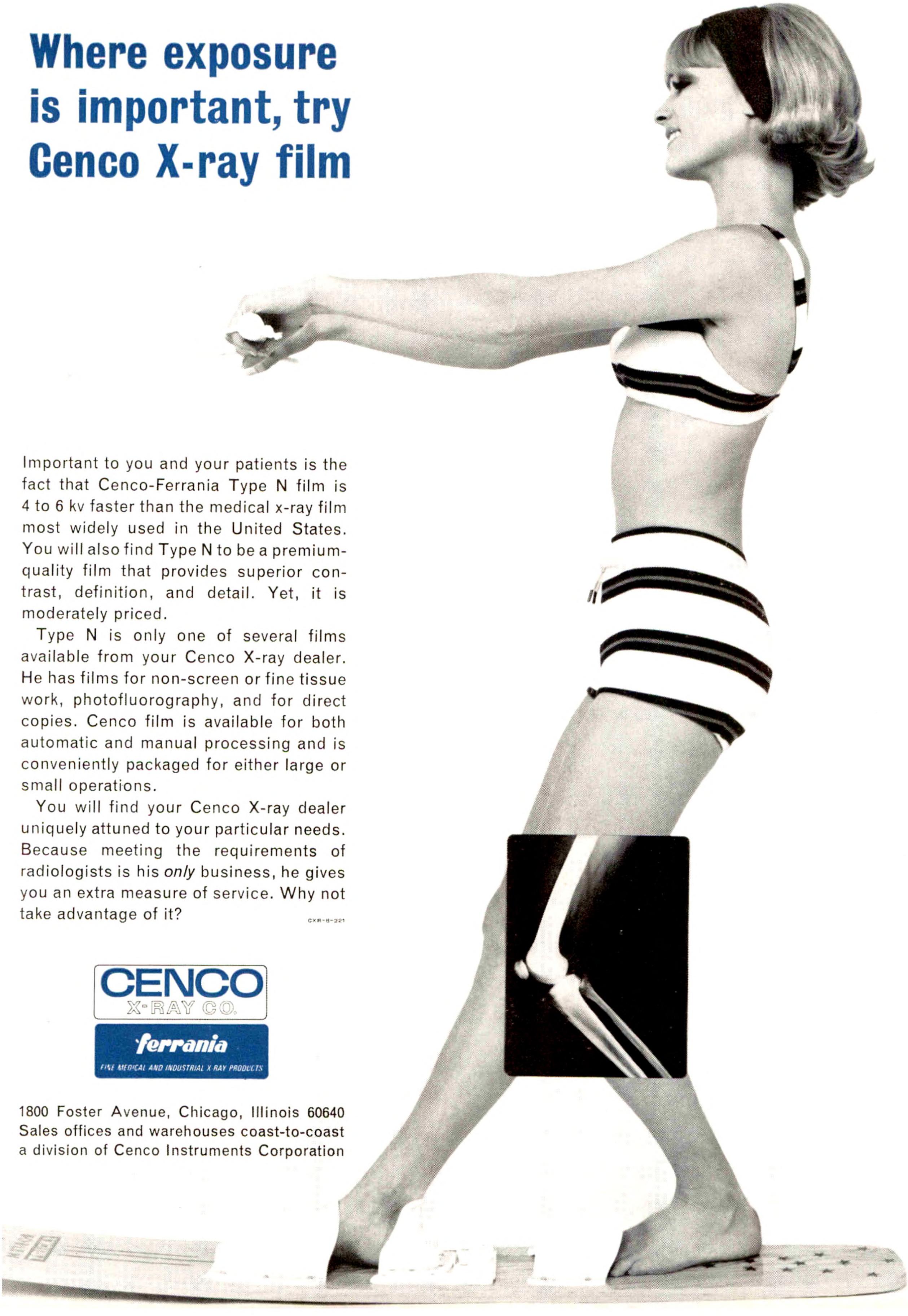

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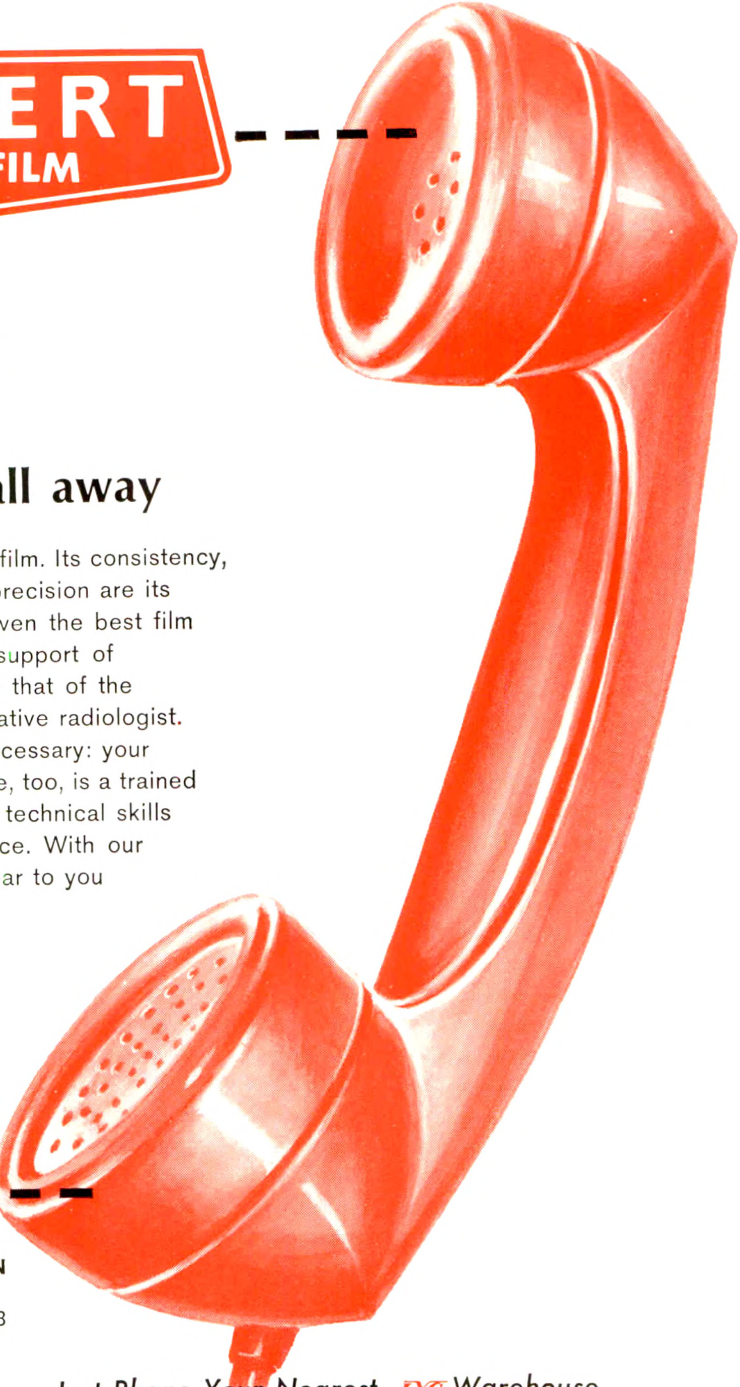

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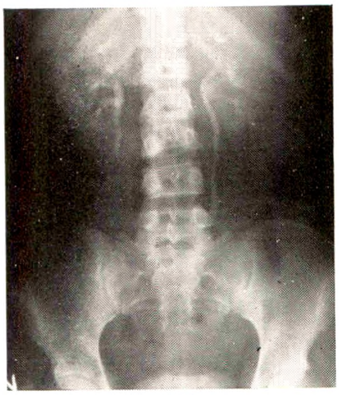
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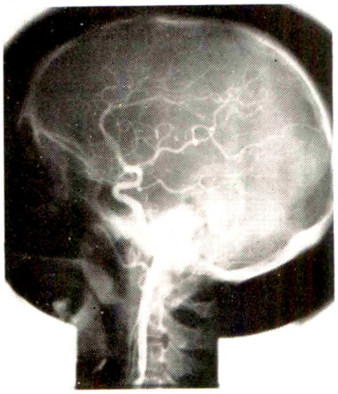
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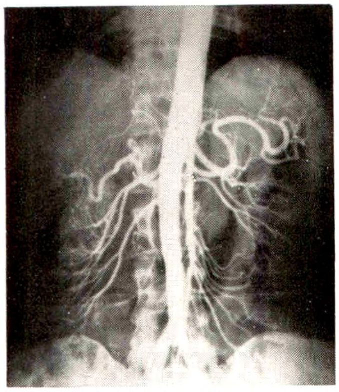
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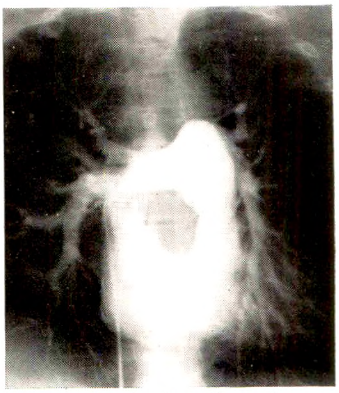
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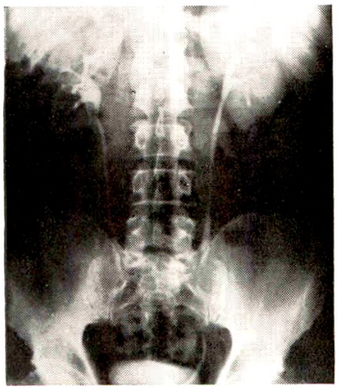
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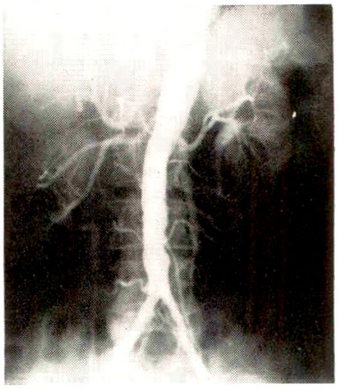
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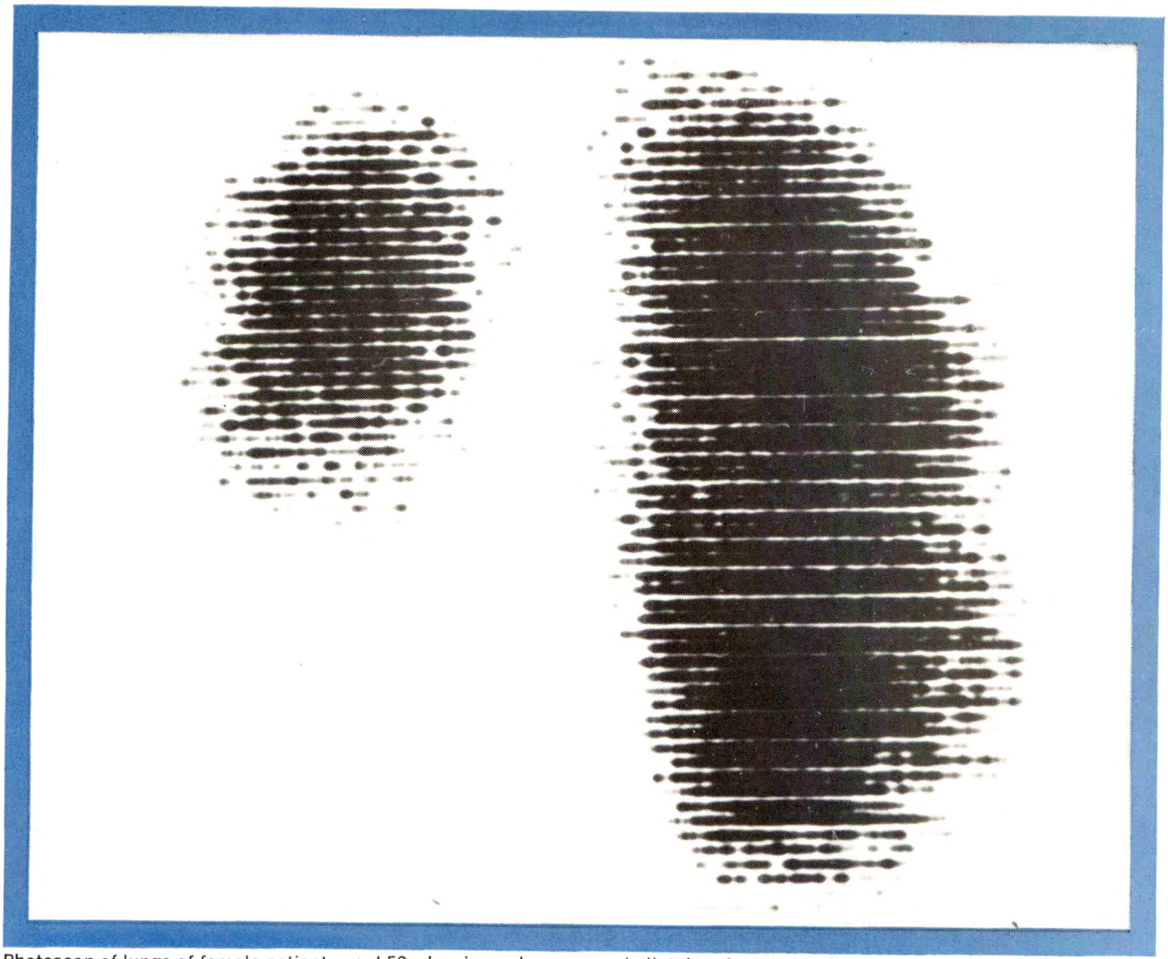

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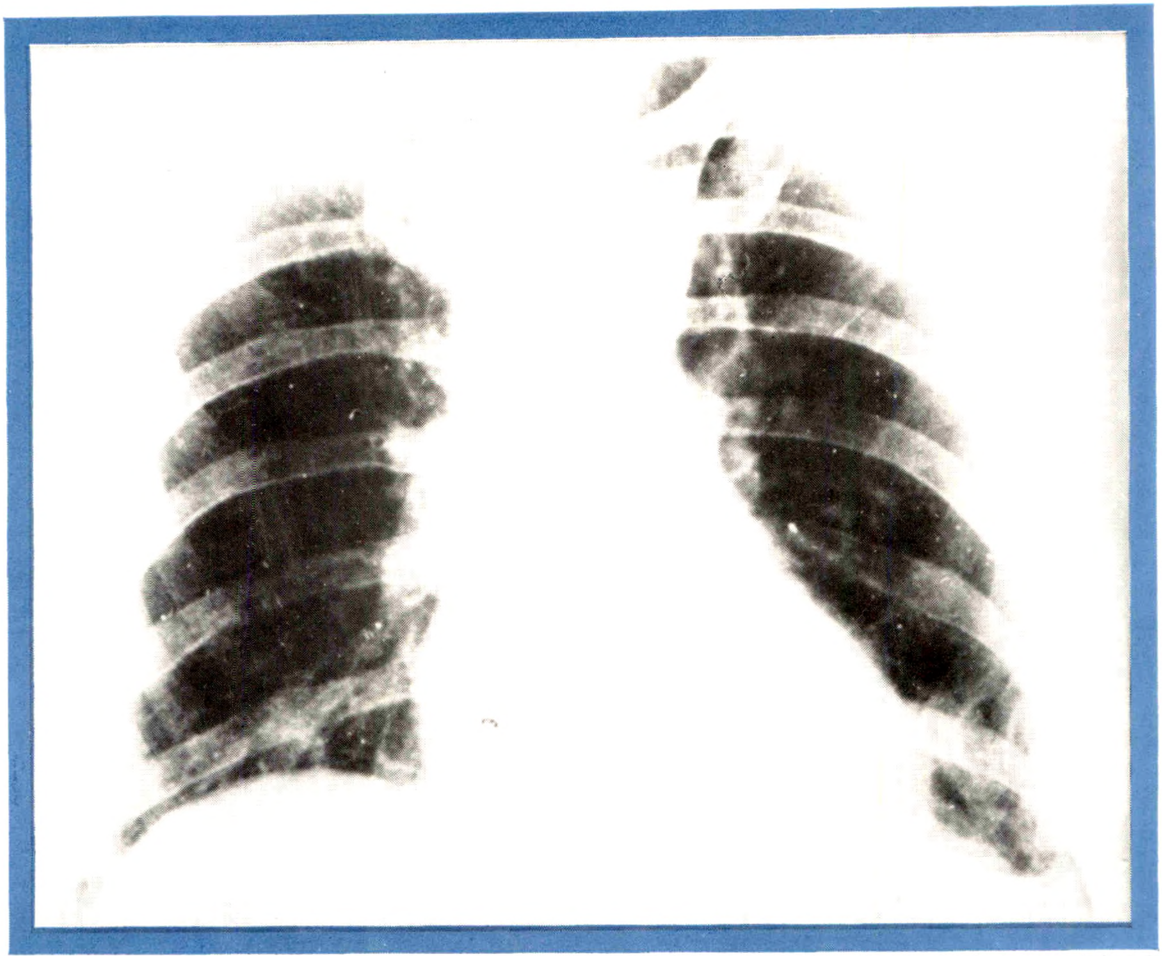

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Dosage and Scanning Procedure: Recommended scan doses of 150 to 300 microcuries of aggregated radioiodinated (I^{131}) albumin depending on the instrumentation available and the technics employed. Scanning immediately follows administration of slow intravenous injection. Patient may be placed in a prone or supine position.

Side Effects and Precautions: There have been no reported cardiovascular or other untoward effects attributable to Albumotope-LS. Extensive clinical use of Albumotope-LS has not borne out the hypothetical possibility that particles of large size might induce deleterious cardiovascular or cerebrovascular effects. The product appears to possess no antigenic properties. One patient with a known history of angioneurotic edema, who had been given Lugol's solution in conjunction with aggregated radioalbumin similar to Albumotope-LS, developed urticaria.

Radioisotopes should not be used in pregnant women, nursing mothers, or in patients under 18 years of age unless indications are very exceptional.

Available: As a sterile, non-pyrogenic, aqueous suspension. Each cc. contains approximately 1 mg. aggregated human serum albumin labeled with 800-1500 microcuries of iodine-131 at time of manufacture. Also contains 0.9% benzyl alcohol as a preservative.

References: (1) Haynie, T. P.; Hendrick, C. K., and Schreiber, M. H.: J. Nucl. Med. *6*:613, 1965. (2) Quinn, J. L., et al.: J. Nucl. Med. *5*:1, 1964. (3) Sabiston, D. C., and Wagner, H. N.: Ann. Surg. *160*:575, 1964.

*Illustrations furnished through the courtesy of George V. Taplin, M.D., Harbor General Hospital, Torrance, California.

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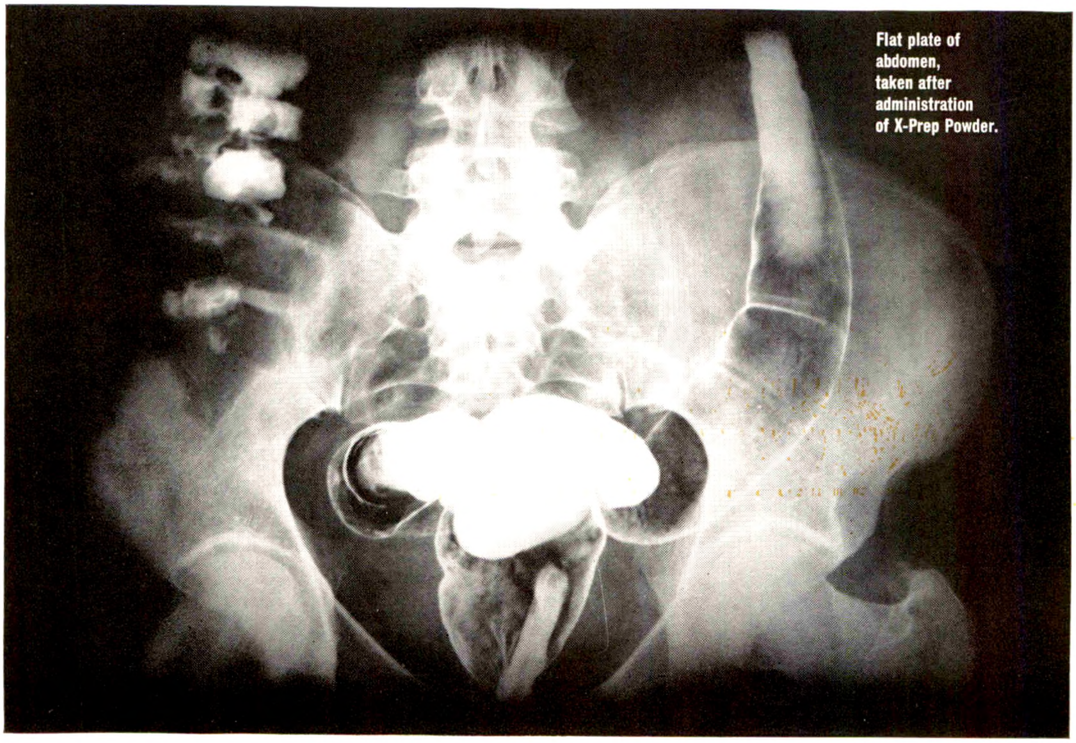
Squibb Quality—
the Priceless Ingredient

NOW…
A ONE-STEP PROCEDURE FOR EFFICIENT CLEANSING OF THE COLON PRIOR TO RADIOGRAPHY



Flat plate of abdomen, taken after administration of X-Prep Powder.

Superior Visualization…Fewer Retakes
with

X-PREP
POWDER
(standardized senna concentrate)
Easy-To-Mix With Water
¾ oz. can
(complete adult dose)

X-PREP
LIQUID
(standardized extract of senna fruit)
Ready-To-Drink
2½ oz. bottle
(complete adult dose)

SIMPLE, PALATABLE, SINGLE-DOSE PROCEDURE FOR GOOD BOWEL PREPARATION
A reliable, predictably effective bowel evacuant that achieves efficient cleansing of the colon by virtually colon-specific peristaltic stimulation. Reduces or eliminates the need for preparatory enemas. Highly palatable and easy-to-take, X-PREP Powder/Liquid encourages better patient cooperation. All the patient has to do is drink the preparation between 2 and 4 P.M. on the day prior to radiography.

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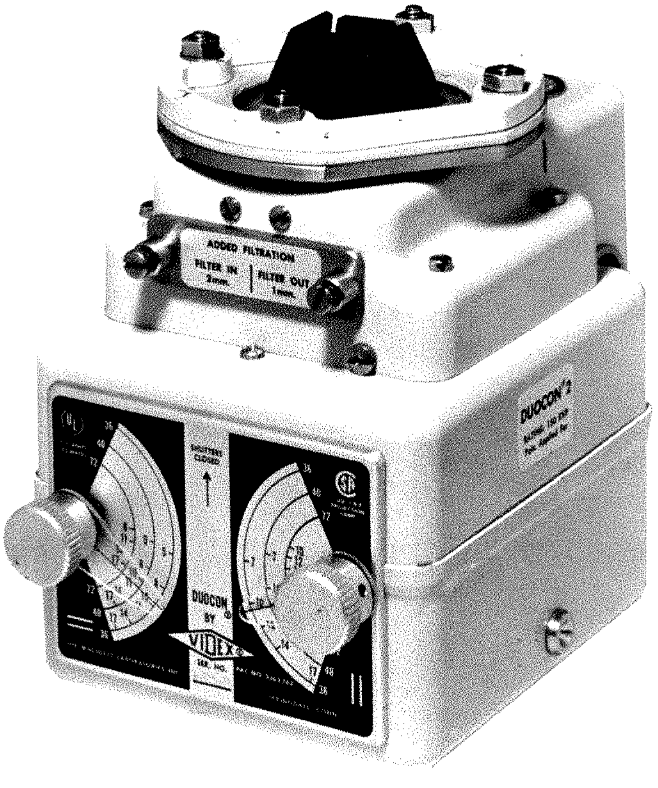
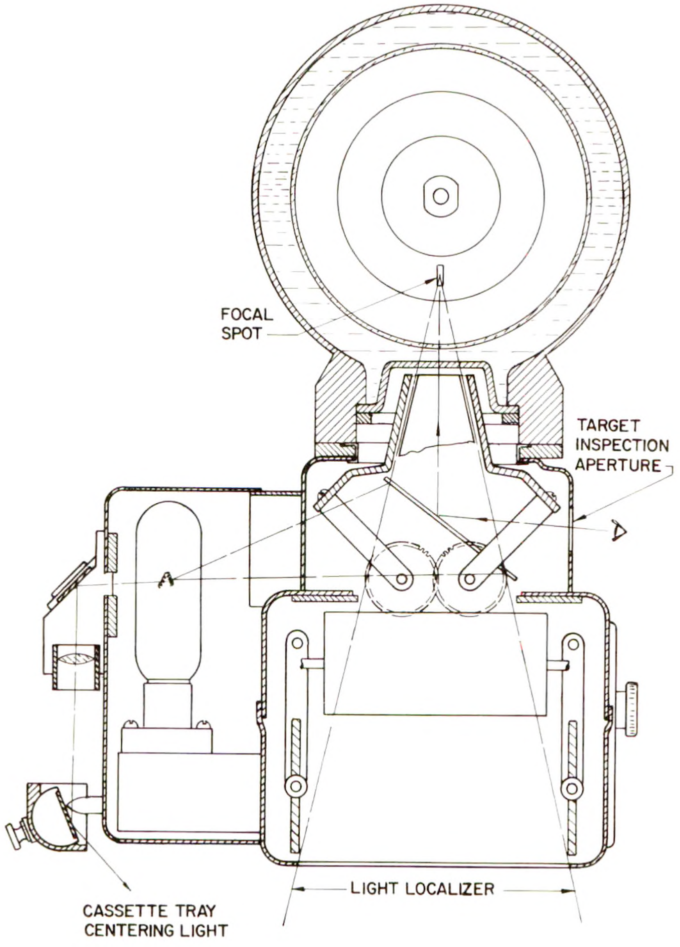
DUOCON-2

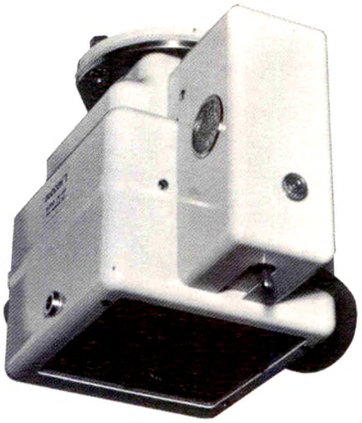
A Compact—Lightweight—Heavy-Duty Collimator

This lightweight Machlett Videx Collimator, the Duocon-2, is designed for heavy-duty use for all radiographic technics, including 150 KV radiography. Special provision for the control of off-focus radiation from the tube is made in the form of a pair of movable upper shutters. These shutters together with other radiation barriers provide well-defined beam localization without weight penalty—the Duocon-2 weighs 60% less than similarly rated multiple-tier collimators.

Following the merger of the Howdon Videx Company into The Machlett Laboratories, Inc., you will find the full line of Duocon radiographic collimators and accessories is now available through Machlett to meet your needs.







DUOCON-2

is easy to operate

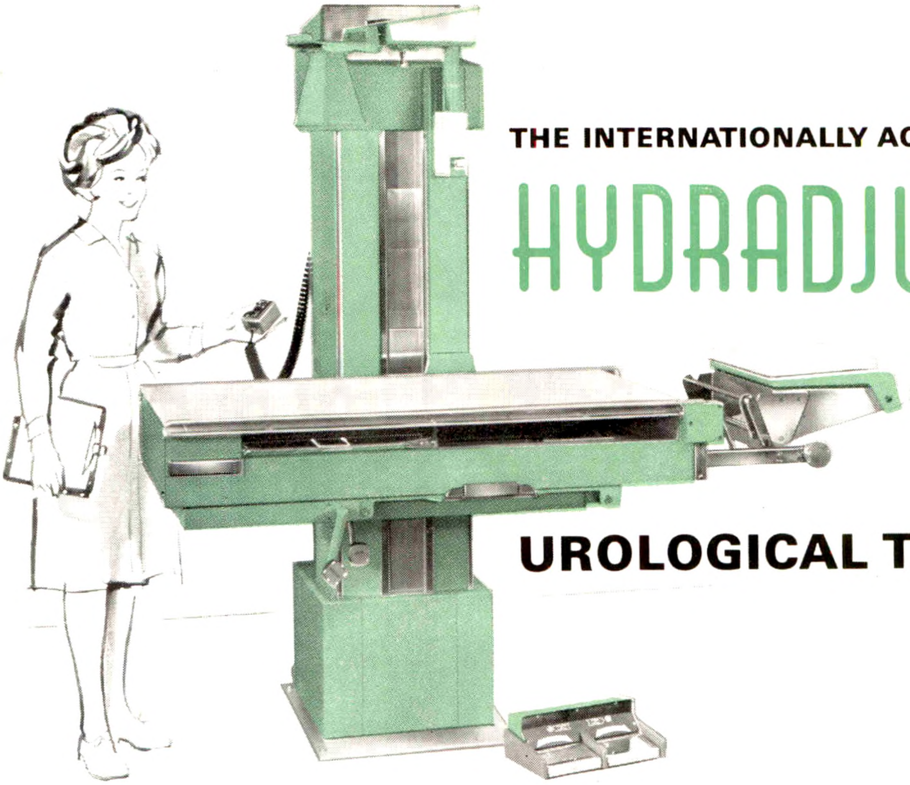
Clear, high-visibility dials, together with easily operable shutter control, permit both accuracy and speed in repeated settings. Duocon-2 provides cassette tray centering light and light localizer for quick, accurate alignment. Localizer lamp is accessible. Mirror viewing system permits observation of X-ray tube target, as illuminated by X-ray tube filament.

Specifications

Rating	150 KVP
Lower long shutter	3.25 mm. lead min.
Lower cross shutter	3.25 mm. lead min. down to 1″ aperture 2.25 mm. lead min. 1″ to 0″ aperture (Note: angle factor varies)
Maximum coverage	17¼″ x 17¼″ @ 36″
Minimum coverage	0″ x 0″
Filtration	Filter in: 2mm equiv. Filter out: 1mm. equiv.
Projected Bucky Tray centering line	Optional extra
Electrical systems Manual	75 watt/115 volt CBX projection lamp, on/off manual toggle switch
Timer (optional extra)	75 watt/115 volt CBX projection lamp, ½ minute automatic switch
Weight on tube stand	7¼ lbs.
Extension cylinders	D-10 and D-10 x 18*
Swiveling	Spring detent

*Assortment of easily attached extensions available, ranging from single 4″ x 10″ to Etter Precision Mastoid Cylinder.

For further details on the Duocon-2 and other Videx collimators, write The Machlett Laboratories, Inc., Springdale, Conn. 06879.

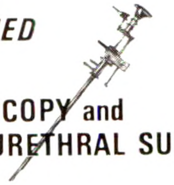


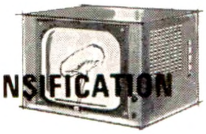
THE INTERNATIONALLY ACCEPTED

HYDRADJUST®

UROLOGICAL TABLE

DESIGNED FOR:





CYSTOSCOPY and TRANSURETHRAL SURGERY . . . RADIOGRAPHY . . . and IMAGE INTENSIFICATION

The Hydradjust Table's outstanding features give you greater operational ease and allow greater diagnostic thoroughness throughout the wide range of urological procedures.

- **CANTILEVER DESIGN.** Permits under-table mounting of image intensification units (9 in. or smaller), allowing you to see motion in many urological procedures which in the past had to be done blindly.
- **HIGHER ELEVATION AND GREATER TILT RANGES.** Elevation range is from 29″ to 57″—tilt from 15° Trendelenburg to 88° upright. Ranges satisfy requirements for all transurethral and urological radiographic procedures.
- **POWER DRIVEN FLOATING TABLE TOP.** (Optional) The speed and effortless motion of the table top are ideal for scanning urinary tract during image intensification and cineradiographic procedures.
- **STAINLESS STEEL DRAIN PAN.** (Optional) May be used alternatively with the standard disposable drain bags supplied with each table.

Hydradjust models are available with Underwriters' Laboratories listing for use in hazardous locations, Class I, Group C. For more details about this outstanding table, see your x-ray equipment supplier or write us for the latest illustrated brochure.

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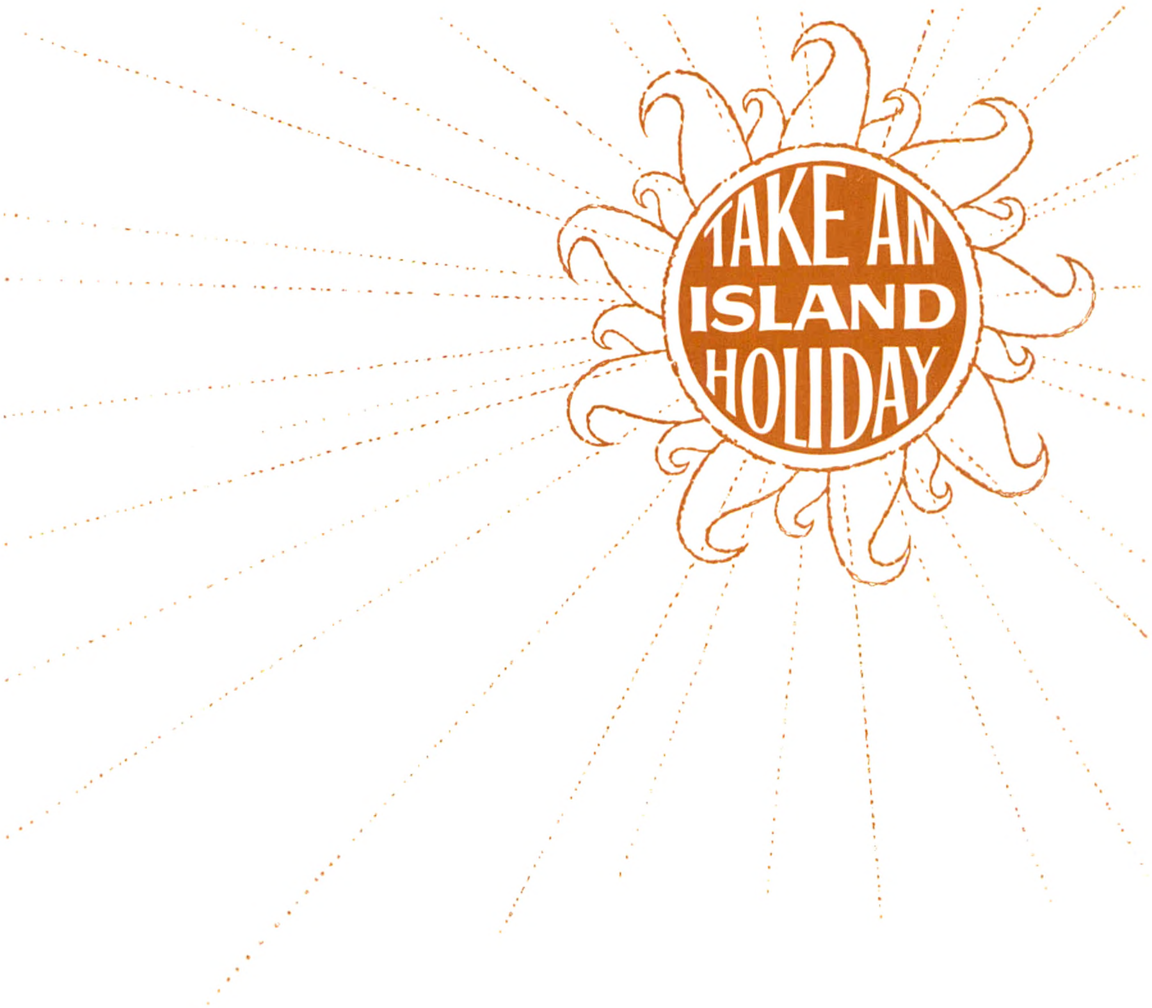
KELEKET



Accessibility: Nothing is quite as easy to get to, get on, get around as an island X-ray table.

Visibility: All around viewing across the board, from either side of the table.

Freedom from fatigue: Spot film tower and tunnel assembly are fully counterbalanced, easily swung into play or parked out of the way for fatigue-free fluoroscopy.

Summing up: Only island units give you this much freedom, this ease of operation, this range of functions, this holiday from the chores associated with any other type of X-ray table. And your only source for true island table design is Keleket, for 65 years, the first name in medical X-ray. Ask for Windsor island tables.

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Medical X-ray Equipment • Accessories and Supplies

Du Pont

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CRONEX X-ray Products

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Announces

New CRONEX* 2 DC, a film designed for the Double Capacity Processing System.

New CRONEX 2 DC, with the same extraordinary diagnostic clarity as today's CRONEX II. Exhaustive tests by Du Pont scientists and by practicing radiologists declare the two films equal in clarity with no change in exposure technic.

New CRONEX 2 DC, made possible through Du Pont emulsion technology and CRONAR® polyester film base. Ideal for Double Capacity processing, CRONAR withstands the heat of high-speed developing, fixing and drying. Because CRONAR is tough it won't tear or jam equipment.

New CRONEX 2 DC, which assures Double Capacity from your present processor, modified as explained in the message on our clear CRONAR sheet. (CRONEX 2 DC works perfectly at normal processing speeds too.)

New CRONEX 2 DC, the film your Du Pont Technical Representative is ready to show you—see him soon.

*Du Pont's registered trademark for its X-ray film.

GRAPH-O-MAT® 90

a chemistry for all times

The choice is yours — where an x-ray department requires extra speed but is unwilling to sacrifice quality, the new **Hunt Double Capacity System** is the answer. Where access time is considered a more important factor, the 90 second dry-to-dry processing cycle will be your decision.

The **Double Capacity System** — engineered and developed by Hunt offers · fast access time, (double conventional film capacity) · films of diagnostic quality comparable to the best quality of the 7 minute cycle · and the choice of using any brand of film; X-ray departments using a particular film emulsion can maintain the high standards established through long association with a favored film.

■ Whatever decision you make — think of **Hunt**. Our chemistry, Graph-O-Mat® 90 is compatible with both processing cycles and all brands of film. Our sales force will offer you knowledgable advice on every aspect of the new ultra-fast processing cycles and our trained staff of service engineers is ready to convert your existing processors.

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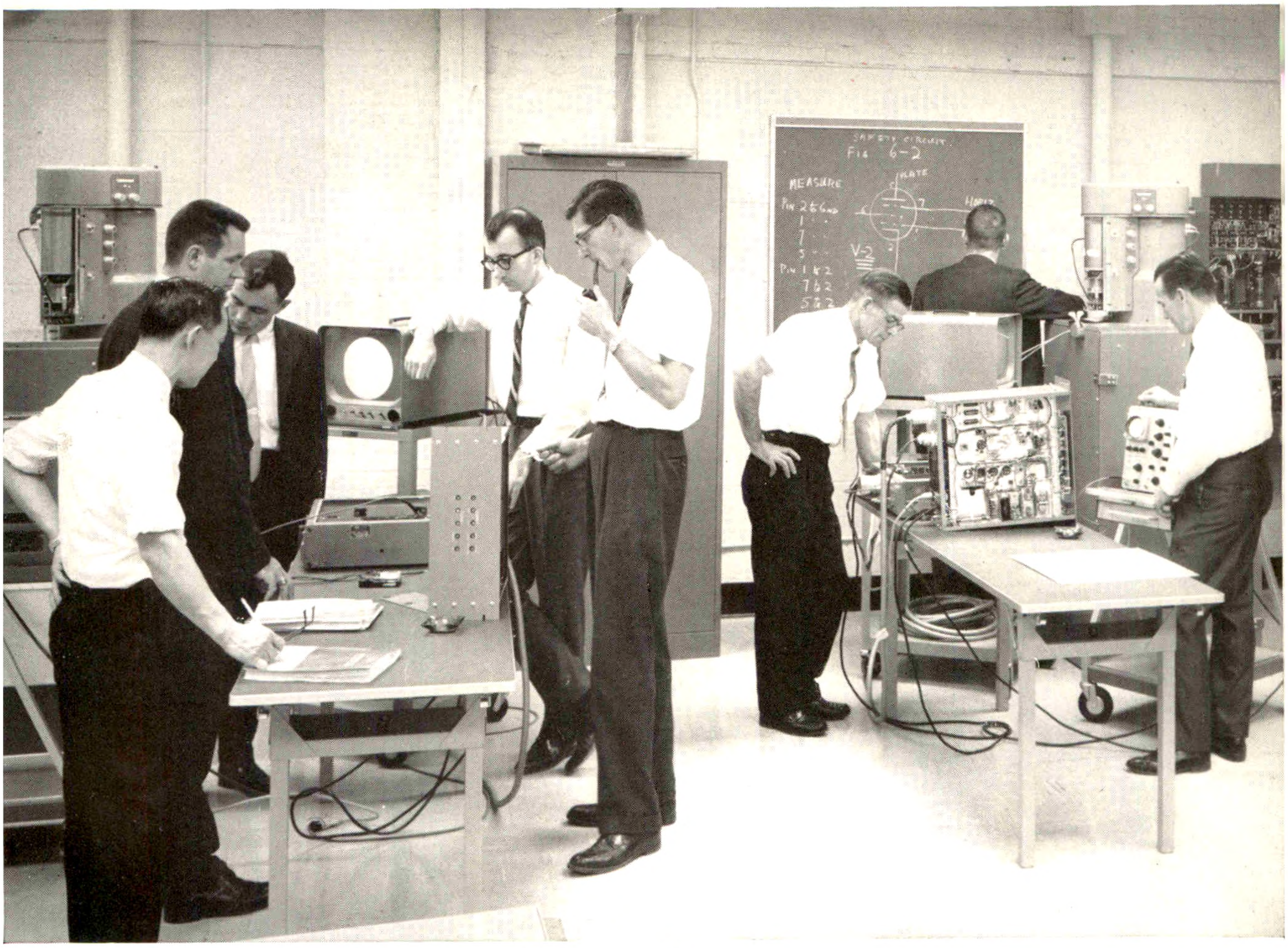

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A Picker service engineer's education is never done.

To keep on top of today's mounting flood of technical developments and equipment innovations demands not only unremitting personal study, but periodic group brush-up in the unique "refinishing" school at our Cleveland plant.

Looking in on a typical week there, you'd find a gathering of service engineers brought in from Picker branches the country over to undergo a week of intensive training.

Not only hard-driving daily classroom instruction by a full-time teaching staff, but expert-guided small group "workshop" sessions dealing with counterparts of the advanced apparatus they'll be servicing back home.

The men pictured above comprise one of four similar separate groups working simultaneously, each occupying an identically equipped corner of the spacious workshop. The program goes on year-round: every member of the vast Picker service organization attends periodically to elevate his competence to serve you.

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People you can bank on for the best in equipment, in supplies, in maintenance.

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Sterile Solution
CYSTOKON™
[SODIUM ACETRIZOATE 30%]
For Cystography
NOT FOR INTRAVASCULAR ADMINISTRATION
Each ml. contains 0.30 Gm. Sodium Acetrizoate,
mg. calcium disodium ethylenediaminetetraacetate
not more than 0.15 mg. sodium biphosphate as a buffer.
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LINKING CHEMISTRY TO MEDICINE

CYSTOKON

the first contrast medium 'tailor-made' for CYSTOGRAPHY

all you add is the infusion set

- premixed—no powders to measure, no media to mix and dilute
- presterilized—no bottles to clean, sterilize or autoclave
- easily administered by gravity flow or other instillation techniques
- new screw-cap bottles—readily adaptable to venoclysis sets

CYSTOKON™

(SODIUM ACETRIZOATE 30%)

Unsurpassed as a safe, efficient, economical contrast agent

Well tolerated by urinary tract mucosa ■ High iodine content for maximum contrast with minimum salt concentration ■ Rapidly diffused in the urine for homogeneous opacification ■ No waste—30-50% lower cost than comparable quantities of other commonly employed agents

Three bottle sizes for more flexible dosage and volume ranges

250 ml. single-dose, disposable bottle (shown at left) ready to use full strength for most cases

500 ml. dilution-unit bottle holding 250 ml. Cystokon ready for dilution to desired concentrations from 30% to 15%

100 ml. multiple-dose vial to provide further concentration and volume flexibility

Caution: Not for intravascular administration

Description: Cystokon is a sterile aqueous solution containing 30% w/v of the sodium salt of 3-acetamido-2,4,6-triiodobenzoic acid. Each ml. contains 0.3 Gm. sodium acetrizoate (0.2 Gm. iodine), not more than 0.05 mg. calcium disodium ethylenediaminetetraacetate as a stabilizer, and not more than 0.15 mg. sodium phosphate as a buffer. The pH of the solution is adjusted to within 7.0 to 7.4. Since the solution itself will not support bacterial growth, additional bacteriostatic agents have not been added. The solution is somewhat sensitive to light and must be protected from strong daylight or direct exposure to the sun.

Clinical Application: Cystokon is prepared and packaged specifically for cystography and retrograde pyelography. It may be employed in simple retrograde cystography and pyelography, voiding cystourethrography, delayed cystography and other appropriate modifications of these techniques.

Contraindications and Side Effects: No evidence of irritation or toxicity to the urinary tract mucosa has been reported with Cystokon. However, minor side reactions associated with the procedure, e.g., nausea, vomiting, pain, salivation and sweating, may be encountered in rare instances. Certain pathologic conditions may, in the judgment of the attending physician, contraindicate retrograde pyelography and cystography.

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□ Barotrast suspends easily—and **stays** in suspension. Coverage is complete...coating is thin, elastic, tenacious...flow is steady, consistent, and columnar. Barotrast is ideal for routinely precise examinations. □ BAROTRAST® (Specially compounded and micronized form of barium sulfate) □

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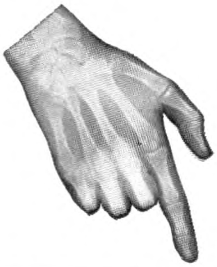
DIVISION OF BARNES-HIND PHARMACEUTICALS, INC.
Sunnyvale, California



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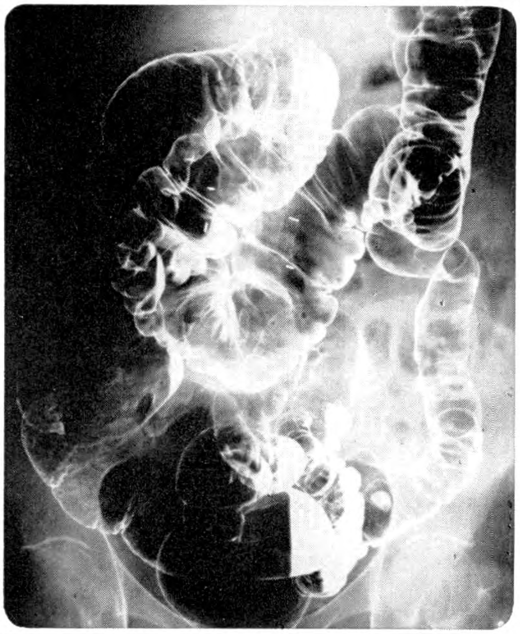
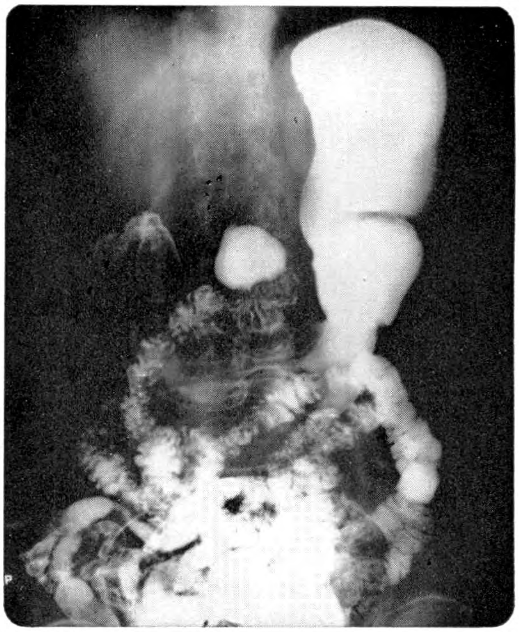


Barotrast used in an air contrast study of the mid transverse colon.



Barotrast used in an upper GI study of the stomach.

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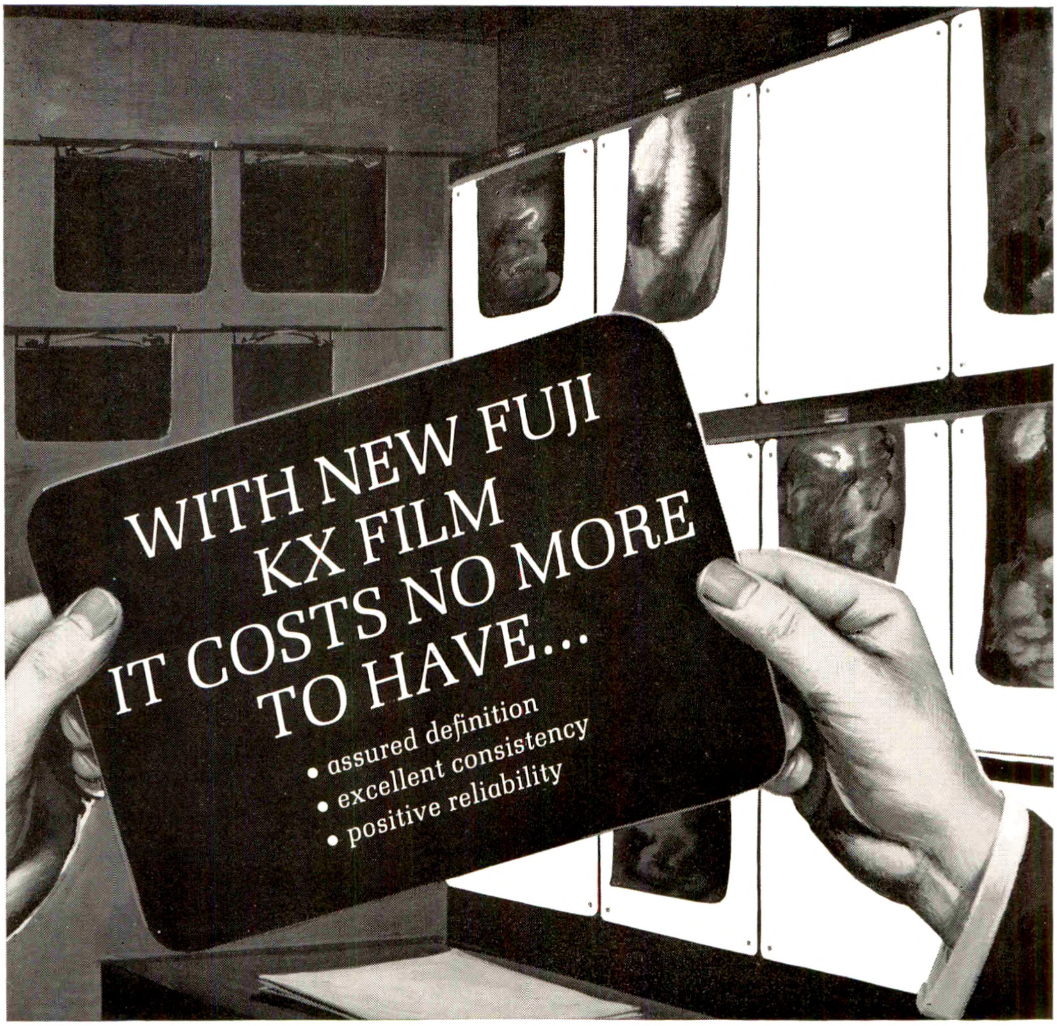

...AND IN MANY CASES AT LOWER COST, TOO!

- compatibility with roller-type processers
- high contrast image
- low fog level
- designed for high KV radiography

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PYNE X-RAY CORPORATION

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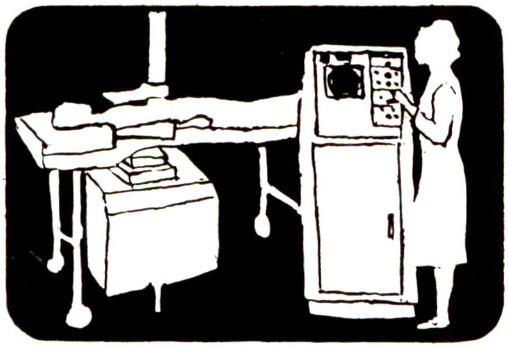
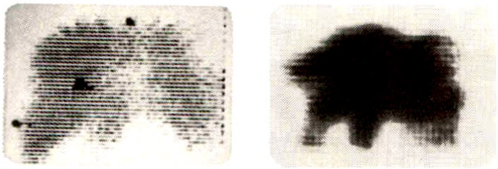
FUJI FILM

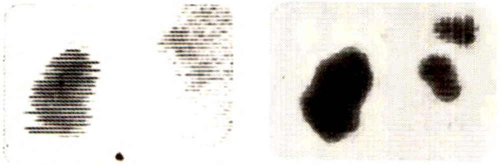
30 YEARS OF SCIENTIFIC ACHIEVEMENT





Conventional Scan Time: 35 min. **Autofluorogram Time: 3.2 min.**

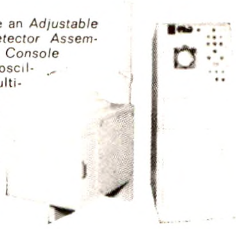
At left is a conventional anterior scan compared with a Model 5000 autofluorogram of an abnormal liver showing a hepatomegaly and an abnormal zone of decreased uptake in the lateral margin of the right lobe, which represents a liver metastasis.



Conventional Scan Time: 20 min. **Autofluorogram Time: 4 min.**

At left is a conventional posterior renal scan compared with a Model 5000 autofluorogram showing a reduced uptake in the inferior 2/3rds of the right kidney due to the presence of a large hypernephroma in this area.

Supplied with each Model 5000 are an *Adjustable Hospital Bed, Motor-Driven Detector Assembly, Recording and Control Console* which includes cathode ray oscilloscope visual display, multi-channel analyzer circuitry with memory, computer-formatted magnetic tape system, and camera mount.



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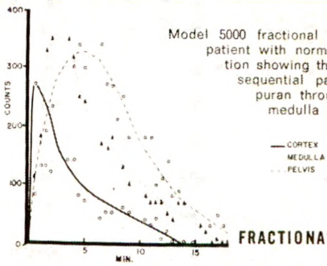
MODEL 5000
DIGITAL AUTOFLUOROSCOPE

FOR RAPID DIAGNOSTIC LOCALIZATION AND VISUALIZATION OF GAMMA-EMITTING RADIOISOTOPES WITHIN THE HUMAN BODY!!



Model 5000 fractional renogram in a patient with normal kidney function showing the characteristic sequential passage of Hippuran through the cortex, medulla and pelvis.

FRACTIONAL RENOGRAM

The Digital Autofluoroscope MODEL 5000 is a fixed (non-scanning) device with memory storage which provides a picture of the distribution of radioisotopes within any organ of the human body. Compared to conventional moving scanners, the versatile new MODEL 5000 will localize tumors faster. Visualization is accomplished by the use of a *multi-crystal matrix* which permits the simultaneous detection of radiation from all parts of the organ or area of interest without scanning. Disease processes in the *brain, heart, lung, kidneys, liver, spleen,* and *pancreas* are routinely detected in *only a fraction of the time* required with conventional mechanical scanning techniques!

Moreover, the MODEL 5000 will *also* measure rapid changes in the concentration of a radioactive material in compartments within the organ. Of special importance, the instrument allows effective utilization of the full range of isotopes — high energy isotopes can now be used and data can be immediately viewed! Further, the Autofluoroscope periodically displays accumulated data allowing observation of buildup. For continuing historical advantage, the patient's records can be permanently stored on magnetic tape for future reference!

MODEL 5000 FEATURES:

- CONTRAST ENHANCEMENT OF PICTURE WITHOUT AFFECTING RAW DATA
- DYNAMIC AND STATIC VISUALIZATION OF THE ISOTOPE WITHIN THE BODY
- FULL RANGE OF (INCLUDING HIGH ENERGY) ISOTOPES CAN BE USED AND ACCUMULATED DATA CAN BE IMMEDIATELY VIEWED
- PATIENT'S RECORDS CAN BE PERMANENTLY STORED ON COMPUTER-FORMATTED MAGNETIC TAPE
- CRYSTAL MATRIX AND MULTI-CHANNEL STORAGE OF ACCUMULATED DATA
- PERIODIC DISPLAY OF DATA DURING BUILDUP PROCESS
- AUTOMATICALLY PROVIDES QUANTIFICATION OF DATA

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Diagnostic X-Ray Table

General Electric designed the complete diagnostic table...then you refined it!

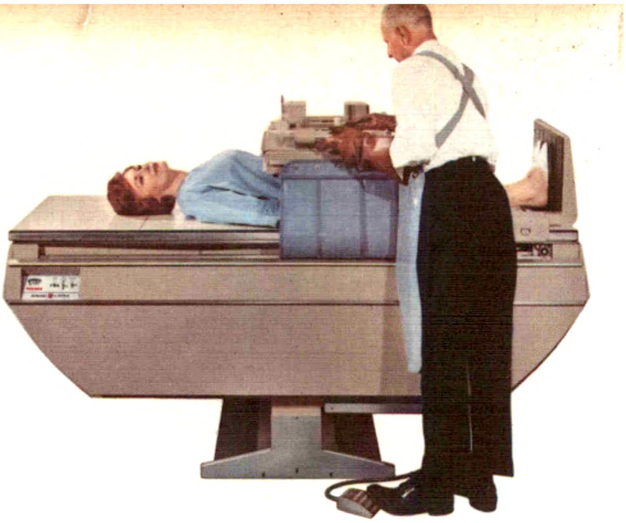
Designed and engineered to perform every radiographic and fluoroscopic function, the Monarch diagnostic x-ray table actually has been refined and improved through *direct hospital and clinical observation*. Our engineers worked with YOU, studying both routine examinations and complex special procedures to meet the demand for a table with responsive performance.

When St. Joseph's Hospital in Milwaukee, Wisconsin selected the Monarch shown above for their new facilities, General Electric personnel worked directly with the hospital and the radiology department from architectural planning t final installation and testing to meet the particula requirements.

When you order a Monarch diagnostic table you're actually ordering an entire installation designed to fit your personal needs. You may equi your table with a wide choice of options—spot film devices, image intensifiers, tube supports Bucky grids and other apparatus, all G-E-designe for maximum operating convenience.

We asked radiologists. We observed every possible procedure. Results...

Patient Safety Features roduced Through Direct Observation



tient comfort . . . operator convenience. No complex radio-ical examination can proceed efficiently if the patient feels ecure or uncomfortable. The Monarch can be equipped th a wide variety of accessories to provide the highest ssible degree of patient comfort and safety plus operator venience. Every device available for the Monarch table s designed after careful investigation of procedural needs. e Monarch diagnostic x-ray table is a direct result of con-uous General Electric product innovation.

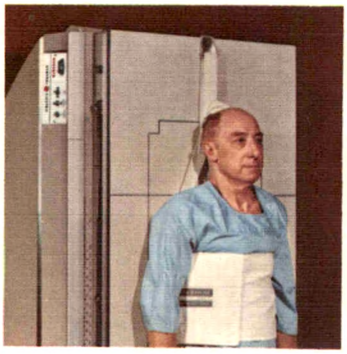
MONARCH

oulder Rest

lipping or sliding of patient in delenburg position. Supports padded with soft polyurethane, correctly spaced for comfort and ndividually adjustable.

Retractable Step

Retractable step assists ambulatory patients onto the table. Eliminates danger of tripping over a separate stool, or having it jam under a power table. Electrical interlock prohibits table angulation when step is extended.



Safety Harness

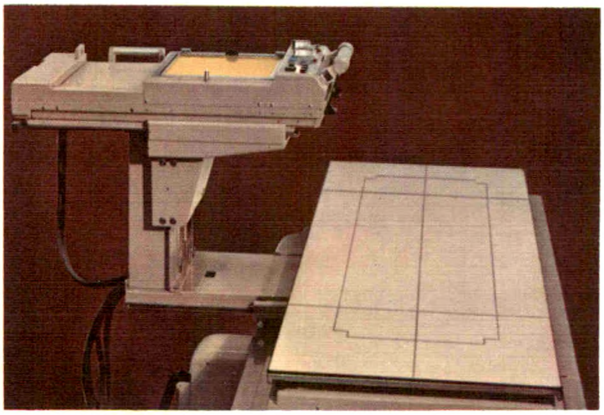
With patients subject to fainting or vertigo, prevent interruption of examination and danger of injury with secure safety harness. Quickly adjustable harness will support 300 pounds.

Grasp Handles

Give the patient a secure feeling and eliminate extra personnel to hold the patient in angulated position. Handles are easily attached or removed at any point on table side. Positive locking device for safety.

Convenient Front-Loading Spot-Film Device

e's the perfect example of how procedural observation and sultation by General Electric engineers results in product ovation. You are able to quickly load the *square cassette* $/_2$" x $9^1/_2$") into the front of the Model 63 spot-film device hout worrying about proper cassette positioning. Carriage chanism is simplified, eliminating gears, belts and pulleys t interfere with spot-film work. And glide-back parking mits table top to be completely cleared during radiographic cedures or for patient care. The square cassette, front load- Model 63 spot-film device is "common-sense" equipment t YOU asked for. We took it from there.



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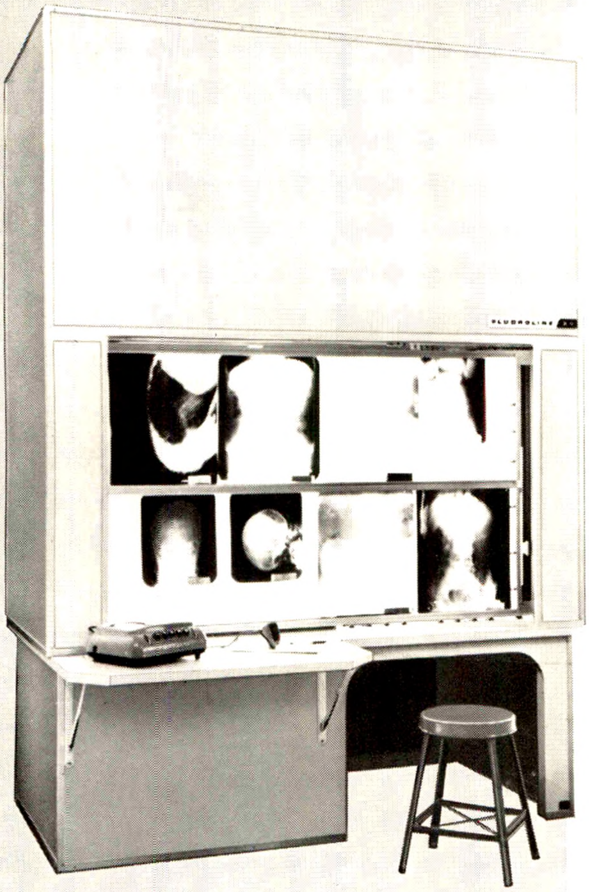
FLUOROLINE® 40-80-160

Multiple Film Viewers



The new Fluoroline film viewers are designed to benefit every member of the x-ray department team. Radiologists can view up to 160 radiographs without time-consuming hand insertion of each radiograph . . . can use their valuable time exclusively for diagnosis. Technicians save time by loading a full series of radiographs at one time, minimizing film handling. Full-function, multiple-panel film viewers have long been in demand . . . now General Electric provides the finest available. Call your G-E x-ray representative or write X-Ray Department, General Electric Co., Milwaukee, Wis. 53201. Room A-61.

FEATURES

- **Manually-Operated Display Panels**
- **Motor-Driven Illuminator Bank With Automatic Safety Stops**
- **Vertical Units Require Minimum Floor Space**
- **Self-Contained Unit**
- **Convenient Front Or Side Shelves**
- **Eight Panel Illuminators (4 Over 4)**
- **Three Film Capacities — 40, 80, 160**
- **Choice Of Vertical, Horizontal, Mobile Or Stationary Models**

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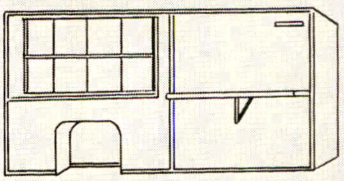

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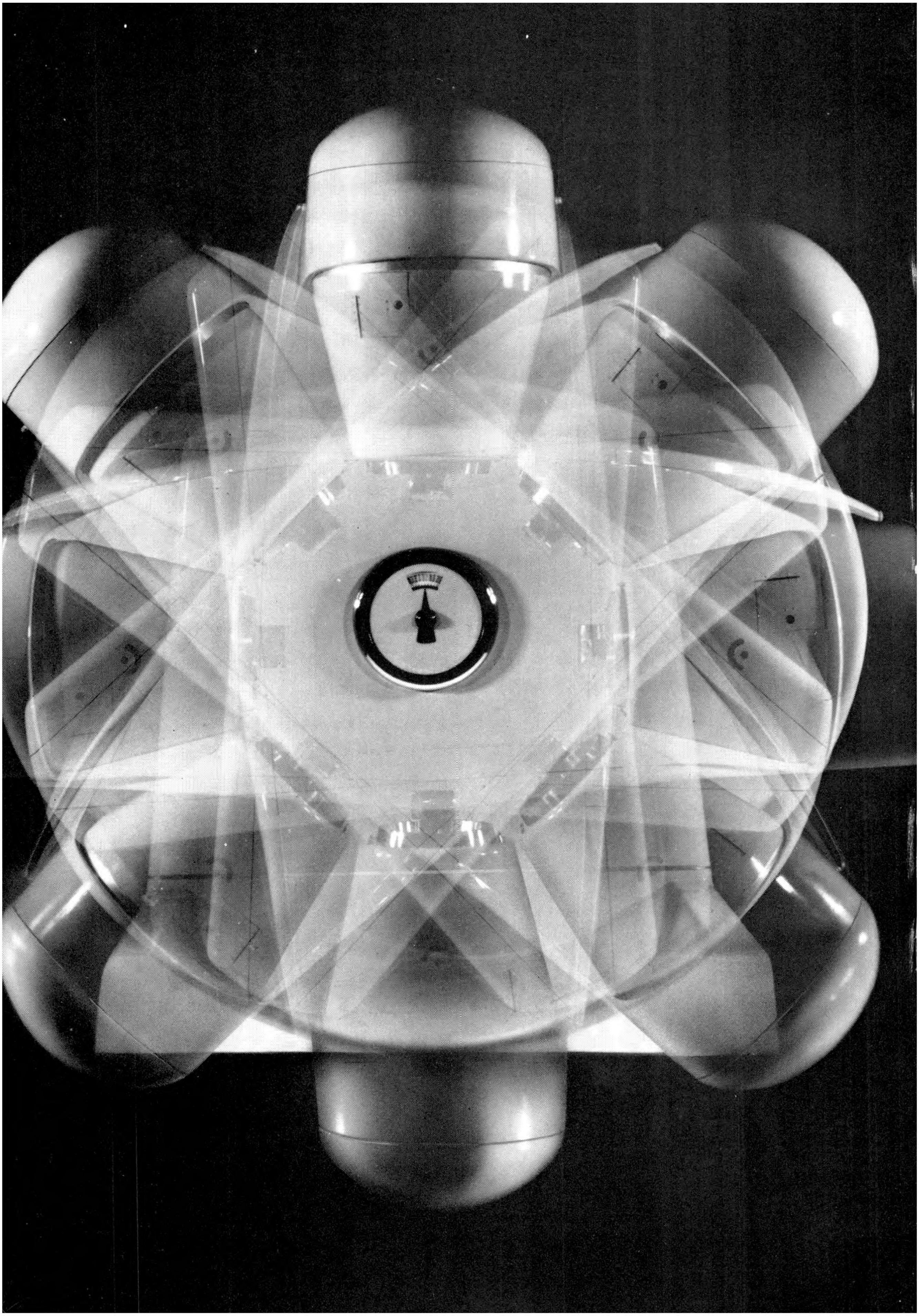

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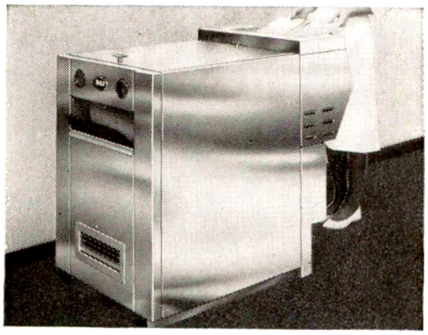

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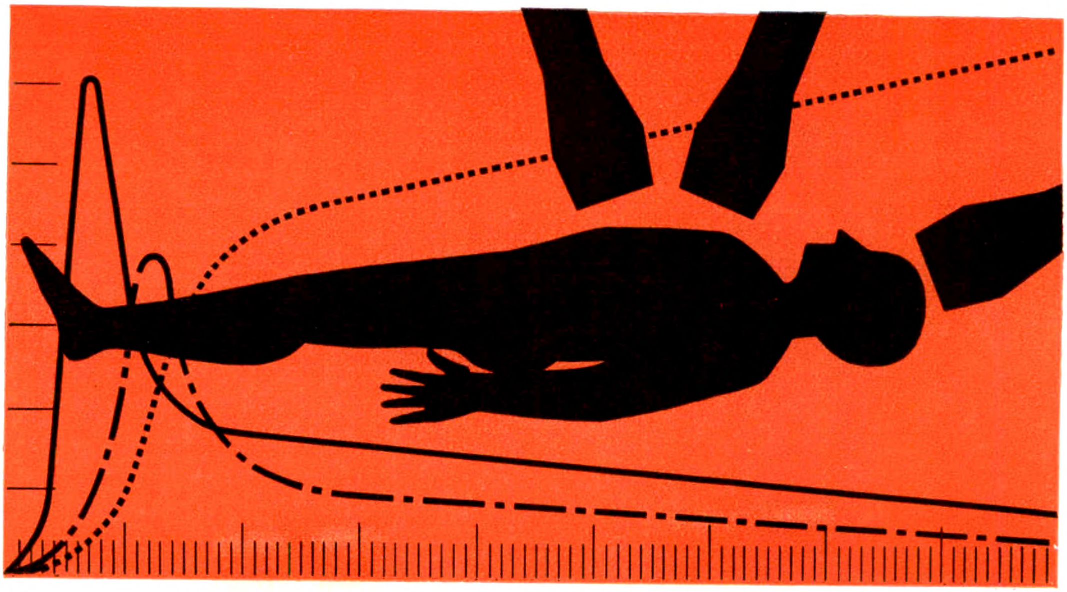

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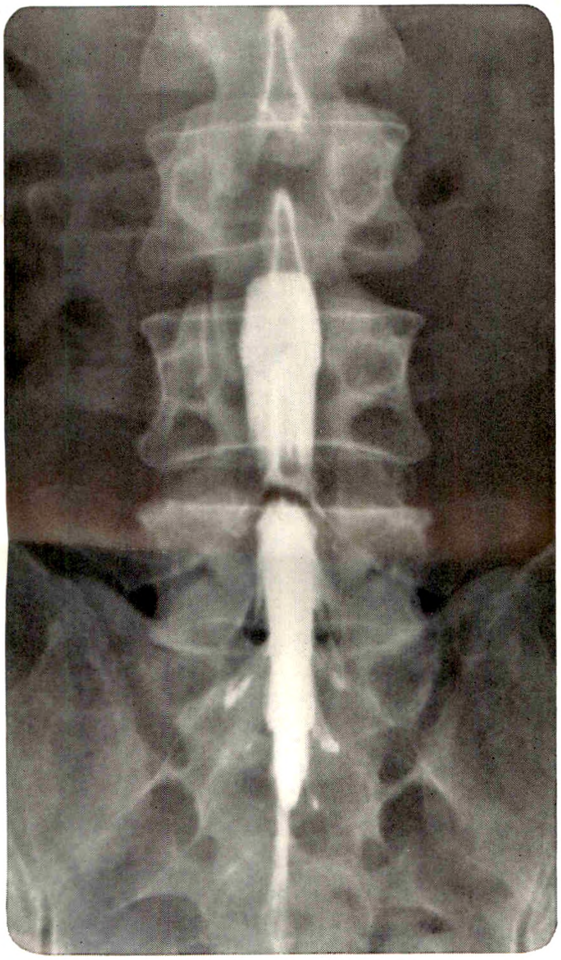

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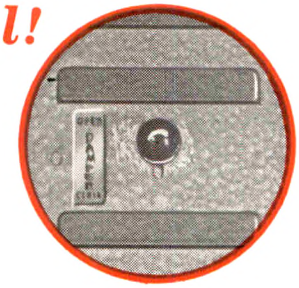

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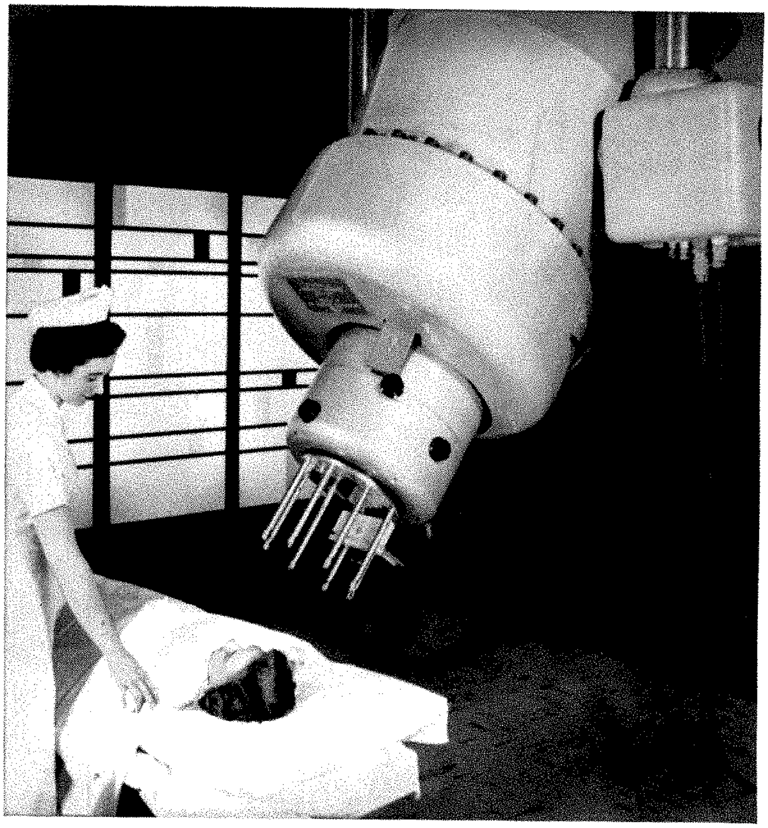

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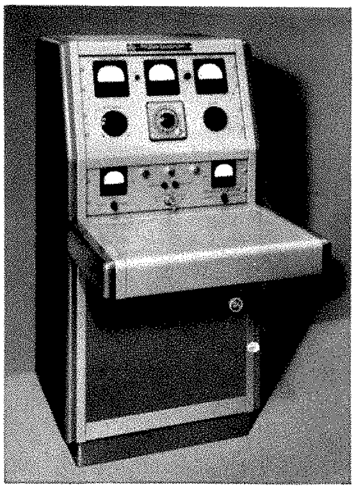

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ANGIOCARDIOGRAPHIC DETERMINATION OF THE SIZE OF THE LEFT VENTRICLE IN CONGENITAL HEART DISEASE*

By AGUSTIN CASTELLANOS, SR., M.D.,† *and* FRANCISCO A. HERNANDEZ, M.D.‡
MIAMI, FLORIDA

PRECISE anatomic diagnosis of left ventricular enlargement is possible by complete roentgenographic and fluoroscopic studies. Cardiologists and radiologists are familiar with the importance of this finding, especially when other procedures, such as electrocardiography, vectorcardiography etc., are being planned. The experienced diagnostician realizes that any of these methods may produce erroneous results in certain cases; mild hypertrophy of the left ventricle is an example. Some left ventricular enlargements are missed and in some instances the diagnosis of enlargement of the left ventricle has proved on follow-up studies to be wrong.

It seemed to us that the end diastolic area of the left ventricle in the anteroposterior angiogram would be a good criterion by which to assess this chamber. To substantiate this belief, we determined in normal individuals the area of this cavity and correlated it with the body surface area (B.S.A.). The values in patients with the most frequently encountered congenital cardiac lesions were determined and compared with the normal. With this method we were able to make correlations with hemodynamic data and we feel that the results are worthy of publication.

MATERIAL AND METHOD

Thirteen normal cases and 75 patients with congenital heart disease were studied. Venous angiocardiograms were made with injection of the contrast medium into the right atrium, right ventricle or pulmonary artery or its branches, using a Cournand

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* From the National Children's Cardiac Hospital, Miami, Florida.

Supported by Research Grant HE-06514-03 of the National Institute of Health, Department of Health, Education, and Welfare, Public Health Service, Bethesda, Maryland; by the Heart Association of Greater Miami; and by the Heart Association of Broward County, Florida.

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catheter No. 5-8 with a NIH tip. Seventy-five per cent hypaque was employed as the contrast medium and 1.0–1.5 ml./kg. was injected for each study with a Cordis injector, the pressures ranging from 500 to 700 lb./inch^2. In most of the cases, a 14 by 14 inch biplane Schönander film changer was used with 4 to 6 exposures per second. Simultaneous recording of the electrocardiogram and of each roentgenographic exposure by means of a Sanborn-Poly-Viso enabled us to correlate each roentgenogram with the time of the cardiac cycle at which it was taken. This device also proved of value in detecting the cardiac arrhythmias sometimes elicited during the procedure.

Correction for distortion due to geometric magnification was made by measuring the actual diameter of the catheter on roentgenograms. We found that the correction was more accurate when the outer diameter of the catheter was measured on the first roentgenograms, in which no contrast material is as yet seen. Our correction figures varied from 10 to 25 per cent, depending on the anteroposterior diameter of the chest, due to the difference in the distance from the thorax to the films, even though the same roentgenographic table was used. The patient was in a supine position on a thick spongy mattress.

The areas of the left ventricular cavities were measured at the end of diastole by means of a planimeter in cm.^{2}. Regular cardiac catheterizations were performed in the majority of the cases prior to angiocardiography. Pulmonary and systemic blood flows were estimated according to Fick's principle.

Approximately 20 minutes before angiocardiography, a test dose of 2 cc. of contast medium was injected intravenously in order to detect any allergic reaction to iodine.

RESULTS (TABLE I)

NORMAL CASES

Thirteen cases considered as "normal" were studied. A direct relationship was found when the end diastolic area of the left ventricle was plotted against the body surface area (B.S.A.) (m.^{2}). The linear regression equation is $Y=a+b\ X=1.1932+(30.1202)\ X\ r=0.992$. Therefore, when multiplying the B.S.A. (m.^{2}) by 30.1, the area of the left ventricle is obtained in diastole in cm.^{2} (Fig. 1).

CONGENITAL HEART LESIONS

The end diastolic area of the left ventricle in 25 cases of ventricular septal defect, 18 cases of atrial septal defect, 20 cases of pulmonary stenosis, and 12 cases of tetralogy of Fallot was plotted against the B.S.A. (m.^{2}) and a direct relationship between both parameters was found to be present.

Table I

SUMMARY OF FINDINGS

	No. of Cases	Age (Yr.)		Body Surface Area (B.S.A.) (m.^{2})		Pulmonary Flow Index (P.F.I.) (L/min./m.^{2})	
		Range	Mean	Range	Mean	Range	Mean
Normal	13	4.5–31	11.1	0.62–1.96	1.17	—	—
Ventricular Septal Defect	25	2.5–25	8.2	0.40–1.80	0.94	2.4–21.0	10.1
Atrial Septal Defect	18	4.0–4.2	10.1	0.63–1.75	1.00	3.0–23.0	11.1
Pulmonary Stenosis	20	4.5–35	13.1	0.62–1.80	1.10	1.7– 6.2	3.5
Tetralogy of Fallot	12	1.5–20	5.7	0.50–1.55	0.73	1.7– 4.4	2.9
Total	88						

Linear regression equations are:

Ventricular septal defect
$Y=a+b\ X=10.707+(29.4268)\ X \quad r=0.657$
Atrial septal defect
$Y=a+b\ X=9.044+(19.4597)\ X\ r=0.827$
Pulmonary stenosis
$Y=a+b\ X=0.343+(26.3112)\ X\ r=0.871$
Tetralogy of Fallot
$Y=a+b\ X=10.6741+(15.0852)\ X\ r=0.854$

Ventricular Septal Defect. Two cases had some degree of mitral insufficiency; 1 had isolated dextrocardia; 1 had high pulmonary resistance; and 2 showed hyperkinetic hypertension combined with some degree of vascular obstruction. Seven cases showed significant differential pressure from the right ventricle to the pulmonary artery due to increased left to right flow and some degree of infundibular stenosis, and 2 had corrected transposition of the great vessels. The remaining cases were isolated ventricular defects and large left to right shunts (Fig. 2).

In Figure 3, it can be seen that the lowest value was obtained in the case with high pulmonary resistance and the highest in one with significant mitral insufficiency. All cases with some degree of pulmonary stenosis showed values above normal. Twenty-two or 88 per cent out of 25 cases showed ventricular areas above the normal regression line.

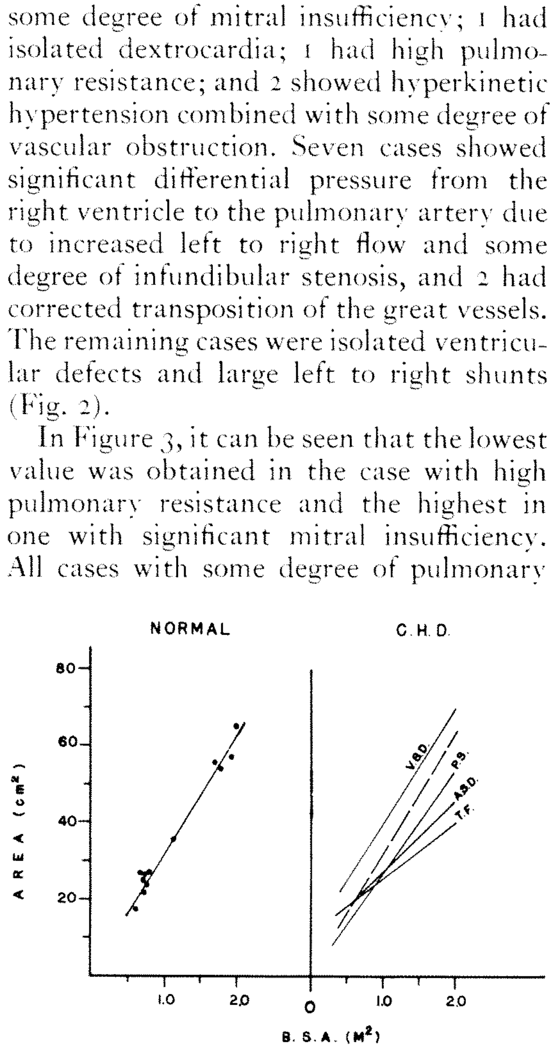

FIG. 1. End diastolic area of the left ventricle plotted against the body surface area (B.S.A.) (m.²) in normal cases and in cases of ventricular septal defect (V.S.D.), atrial septal defect (A.S.D.), pulmonary stenosis (P.S.) and tetralogy of Fallot (T.F.). *Left:* Normal regression line. *Right:* Broken line—normal regression line; solid lines—regression lines of the different groups of congenital heart disease (C.H.D.) cases.

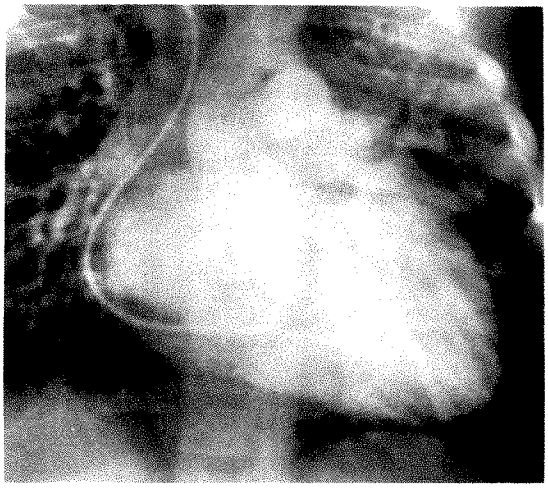

FIG. 2. Ventricular septal defect demonstrated on frontal levo-angiocardiogram at the end of ventricular diastole. Enlarged ventricular cavity. Body surface area (B.S.A.): 1.41 m.². Area of the left ventricular cavity: 71.2 cm.². Pulmonary flow index (P.F.I.): 17 L/min./m.². Left atrial diastolic area is also enlarged. Mitral lesion is not present.

Atrial Septal Defect. This group contained only 1 case with high pulmonary resistance. Eight cases had various types of anomalous drainage of the pulmonary veins (partial).

Figure 3 shows that a case with high pulmonary resistance had a left ventricular area slightly above the normal. Forty-four per cent of the cases showed ventricular areas slightly above the normal regression line (Fig. 4).

Pulmonary Stenosis. There were 4 cases of infundibular pulmonary stenosis; the remaining were valvular. One case was complicated by anomalous drainage of the upper left pulmonary vein. Patent foramen ovale was present in 5 patients. Figure 5 shows that only 6 patients (30 per cent) had areas above the normal regression line; the majority had areas near the line.

Tetralogy of Fallot. Three cases demonstrated a right sided aorta, 1 had a huge aneurysm of the right main pulmonary artery, 1 had marked coarctation of the left

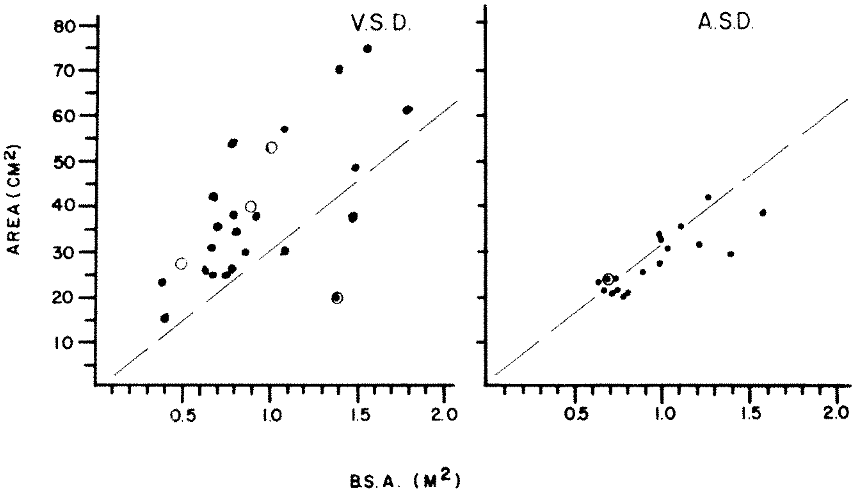

Fig. 3. Plotting of end diastolic area of the left ventricle against the body surface area (B.S.A.). *Left:* In ventricular septal defect (V.S.D.). *Right:* In atrial septal defect (A.S.D.). Broken line—normal regression line. A direct relationship is seen between the parameters in the two cardiac malformations. ⊙ = high pulmonary resistance. Circles = increased flow with increased pulmonary vascular resistance.

main pulmonary artery, 1 had a small patent ductus arteriosus, and 1 case underwent a Taussig Blalock operation on the right side but the anastomosis was not functioning when the angiocardiography was performed. One case showed a left superior vena cava entering the coronary sinus, and 1 presented a large atrial septal defect (pentalogy).

Figure 5 shows that 58 per cent of the cases had ventricular areas which were at the normal or above the normal regression line. Three per cent of the cases were very near to the normal line. At least 25 cases were discarded because of extremely faint opacification of the left ventricular cavities, consistent with very low pulmonary blood flow.[1]

A comparison of the normal regression line with that of each group of cardiac lesions studied, when plotting the end diastolic area of the left ventricle against the body surface area (B.S.A.) (m.²), is shown on the right side in Figure 1. It may be noted that the regression line in cases of ventricular septal defect is above the normal one, regardless of the value of the B.S.A. In order of frequency, below the normal regression line are the lines of pul-

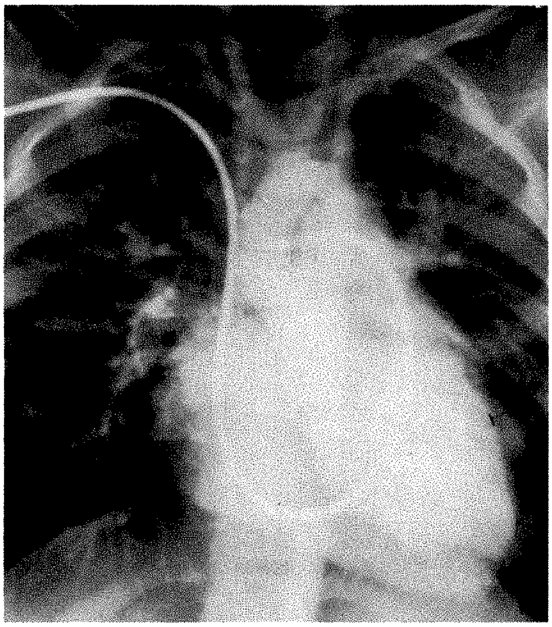

Fig. 4. Atrial septal defect demonstrated on levo-angiocardiogram at the end of ventricular diastole. There is opacification of the right and left cardiac cavities and of the great vessels. The left ventricular area is within normal limits. Note the enlargement of the right ventricular cavity. Body surface area (B.S.A.): 0.99 m.². Area of the left ventricular cavity: 32 cm.²; normal value: 31 cm.².

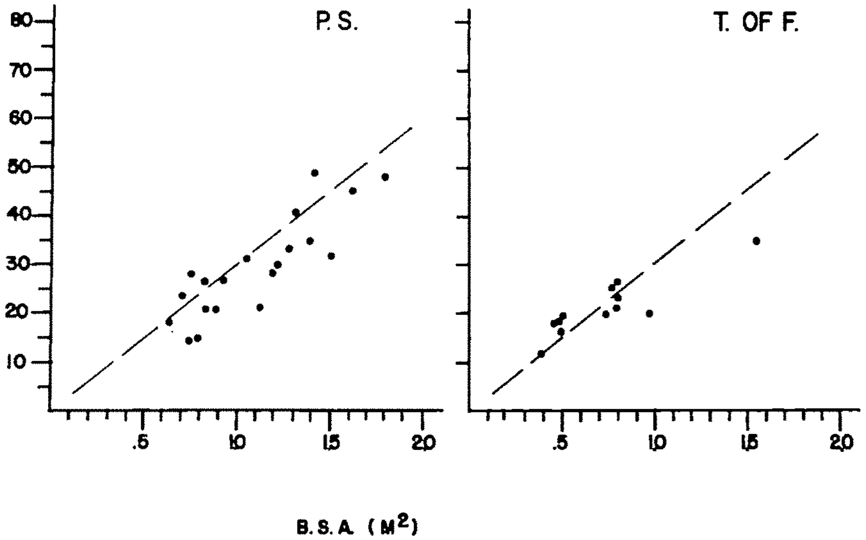

Fig. 5. Plotting of end diastolic area of the left ventricle against the body surface area (B.S.A.). *Left:* In pulmonary stenosis (P.S.). *Right:* In tetralogy of Fallot (T. of F.). Broken line—normal regression line.

monary stenosis, atrial septal defect and tetralogy of Fallot.

RELATIONSHIP OF PULMONARY BLOOD FLOW (P.B.F.) AND PULMONARY FLOW INDEX (P.F.I.) TO THE SIZE OF THE LEFT VENTRICLE

The end diastolic area of the left ventricle in each group of congenital cardiac lesions was plotted against the pulmonary flow index (P.F.I.) (L/min./m.^{2}). A significant direct relationship was found in ventricular septal defect (Fig. 6*A*); no significant relationship was found in atrial septal defect (Fig. 6*B*); and the relationship was inverse in pulmonary stenosis and in tetralogy of Fallot (Fig. 6, *C* and *D*). The regression lines of these two latter groups, if drawn, would be extremely steep. This conclusion is false. In these cases of restricted pulmonary circulation, the greater

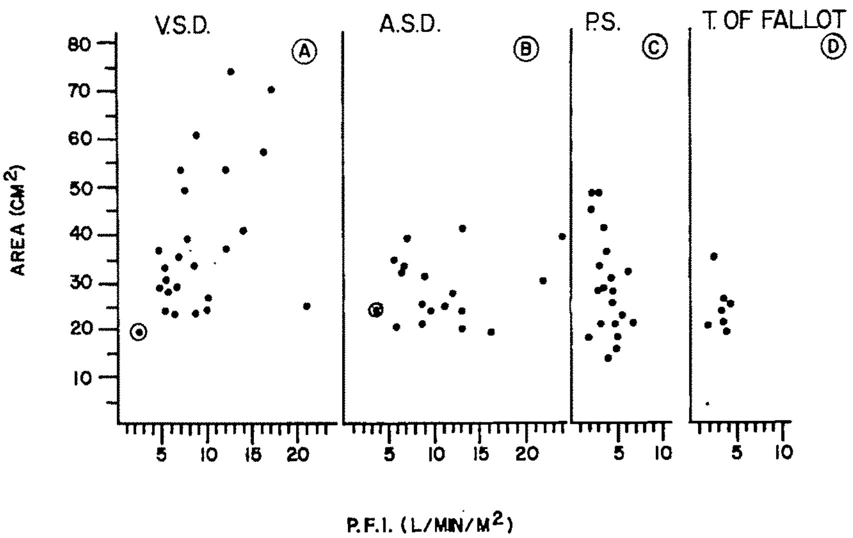

Fig. 6. (*A–D*) Plotting of end diastolic area of the left ventricle against the pulmonary flow index (P.F.I.) (L/min./m.^{2}) in the 4 groups of cardiac lesions considered. An inverse relationship appears to be present in pulmonary stenosis and tetralogy of Fallot. ⊙ = high pulmonary resistance.

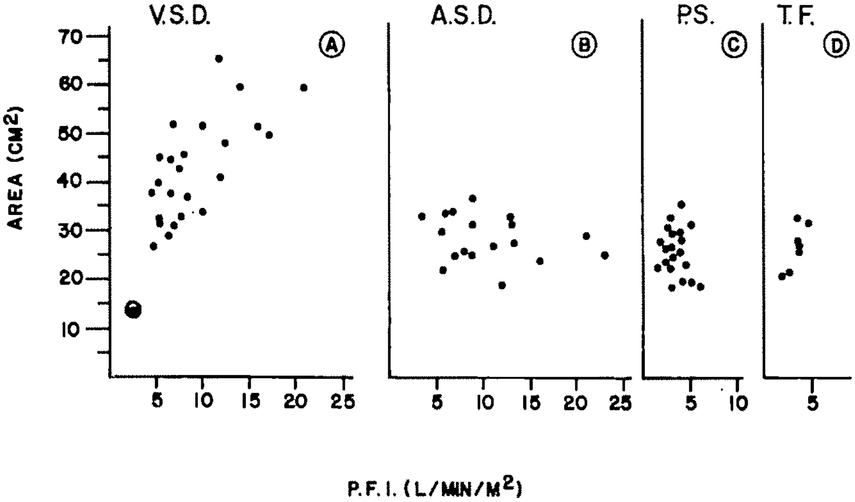

Fig. 7. (*A–D*) Plotting of end diastolic area of the left ventricle, corrected for body surface area (cm.²/m.²) against the pulmonary flow index (P.F.I.) (L/min./m.²) in the 4 groups of congenital heart diseases studied. A direct relationship appears to be present in all of these groups between both parameters except in atrial septal defect in which an inverse correlation is found. ⊙ = high pulmonary resistance.

the flow, the larger the cavity of the left ventricle—the opposite of the result found in this study. Thus, this correlation is not reliable.

The pulmonary flow index (P.F.I.) (L/min./m.²) was plotted against the size of the left ventricle corrected for the body surface area (B.S.A.) (cm.²/m.²). Figure 7, *A–D*, shows that a direct correlation was found in ventricular septal defect, pulmonary stenosis and tetralogy of Fallot but the relationship was inverse in atrial septal defect. This means that the larger the P.F.I., the smaller the left ventricular cavity, although if the regression line were drawn, this would be near the horizontal line. This result is as expected.

When the pulmonary blood flow (P.B.F.)

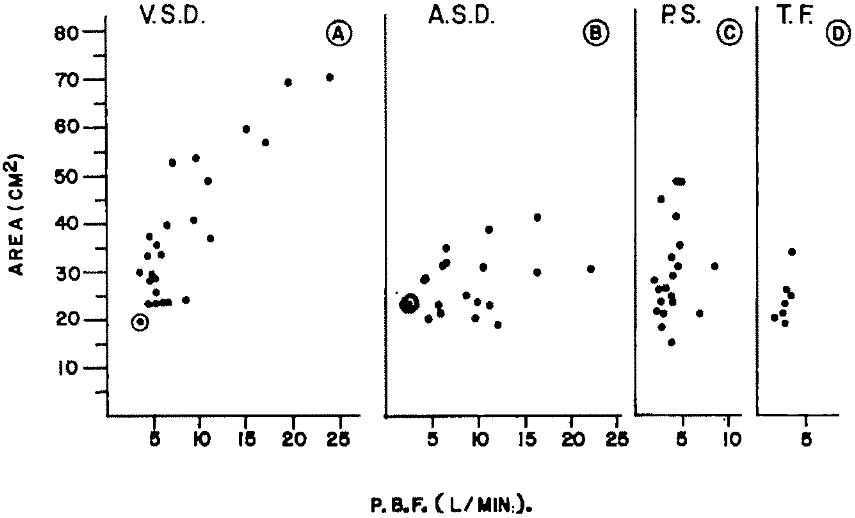

Fig. 8. (*A–D*) Plotting of end diastolic area of the left ventricle against the pulmonary blood flow (P.B.F.) (L/min.) in the 4 groups of congenital heart diseases studied. A direct relationship appears to be present in all of these groups between both parameters, even though much less evident in atrial septal defect. ⊙ = high pulmonary resistance.

(L/min.) was plotted, a direct relationship was found to be present in the 4 groups of cardiac lesions considered (Fig. 8, *A–D*).

DISCUSSION

The determination of the presence or absence of left ventricular enlargement is necessary for the diagnosis of certain congenital and acquired cardiac diseases. Classically, the behavior of the left ventricle is similar to that of the left atrium in cases of patent ductus arteriosus and ventricular septal defect, in which enlargement of the left ventricular cavity has been considered a common finding.[3,6,8,12,13,16,17,19,20,21] Nevertheless, although well known, the small size of the left ventricular cavity in cases of atrial septal defect is rarely emphasized. Also, in mitral and aortic diseases, the assessment of the anatomic condition of the left ventricle is an important aid in the diagnosis.

To date, roentgenographic examination by fluoroscopy and plain roentgenography, primarily in the left anterior oblique position, and delineation of the esophagus by opaque barium mixtures have been used in the determination of the size of the left ventricle. However, in some situations, a diagnosis of slight or moderate enlargement of the left ventricle cannot be accurately made. Laminagraphy has also been tried but has not been found to be of additional value. So far, levo-angiocardiography or left ventriculography, because of the resultant dense opacification of the left ventricular cavity, appears to be the best method for the assessment of the size of this cardiac chamber.

ANGIOGRAPHIC PROCEDURES

Three different angiocardiographic procedures may be utilized in the evaluation of the left ventricle: (1) the estimation of the volume or capacity of this cavity in systole and diastole; (2) the measurement of the transverse and longitudinal diameter of the cavity in two projections, anteroposterior and lateral or both oblique; and (3) the measurement of the area in cm.^{2} in end systole and end diastole in anteroposterior projection.

The first procedure requires bi-plane studies. Accurate mathematical formulas have been developed for frontal and lateral projection and also for both oblique positions. The capacity or volume of the left ventricle[2] is expressed in ml., using a rapid film changer and cine-angiocardiography for this purpose.[1,2,7,9,10,11,15] Another formula has been devised for the measurement of the left atrial cavity,[2] which gives very informative results. The objections, however, are: (a) that the cavity of the left ventricle may be quite different from the geometric configuration to which the mathematical formula is applied; and (b) that very expensive equipment is needed.

Measurement of the transverse and longitudinal diameters is a practical way by which to estimate the size of the cardiac chamber.[14] Nevertheless, it is not reliable when the configuration of the cavity is bizarre or unusual.

Measurement of the end diastolic area of the left ventricle in cm.^{2} gives a "dimension" of greater value than the diameters. When this area is larger than normal, the volume or capacity of the left ventricle is expected to exceed the normal value as well. On the other hand, a small area of the left ventricle in frontal projection means that the capacity or volume is below normal. In other words, there is a direct relationship between the area in frontal projection and the volume or capacity, expressed by a figure (in cm.^{2}), which can be used to accurately assess the size of the left ventricle and compare it with the normal value. The approximate end diastolic area of the left ventricle in normal individuals is obtained by multiplying the B.S.A. (m.^{2}) by the factor 30.1 cm.^{2}. Prior studies[5] showed that the end diastolic area of the left atrium in normal persons can be obtained by multiplying the B.S.A. (m.^{2}) by the factor 23.5; thus, the area of the left atrium is smaller than the area of the left ventricle. With this in mind, in all cases in which the end diastolic area of the left atrium is significantly greater than the end diastolic area of the left ventricle, the presence of mitral disease or other cardiac abnormality should be suspected.

LIMITATIONS OF ANGIOGRAPHY

In the estimation of the volume of the left ventricle by angiocardiography, there are some limitations. The displacement of the heart due to mediastinal masses, pleural effusion, etc., an abnormal position of the heart as in agenesis of one lung, extreme rotation of the heart, etc., render the method inapplicable. In cases of very large left to right shunt at the atrial level, it is sometimes difficult to delineate sharply the outline of the left ventricular cavity. In cases of restricted pulmonary circulation (pulmonary stenosis and tetralogy of Fallot), the opacification of the left ventricular cavity in the levo-angiocardiogram is very faint because of the very low pulmonary flow. The injection should be made into the main pulmonary artery or into the outflow tract of the right ventricle to obtain denser opacification of this cavity.

COMMENT

In our series, 84 per cent of the cases of ventricular septal defect, 44 per cent of those with an atrial septal defect, 33 per cent with pulmonary stenosis and 58 per cent with tetralogy of Fallot were found to have above normal values. In addition to the regression line, however, the value of "*r*" should be kept in mind. The percentage of studies showing above or below normal regression lines is not an accurate evaluation of the size of this cavity in each group of congenital cardiac lesions. In all groups, a variable number of regression lines were so near the normal that they could be considered as normal instead of slightly above or below the normal.

Some findings are of special interest. Unless a small defect is present, all uncomplicated cases of ventricular septal defect should be assumed as being associated with a large left ventricular cavity. Conversely, atrial septal defect is accompanied by a small left ventricular cavity. Pulmonary stenosis and tetralogy of Fallot are generally associated with a normal sized or small left ventricular cavity. Findings which differ markedly from these should alert the radiologist to the possible presence of anatomic abnormalities of the internal structure of the heart or the pulmonary vascular tree, or to other peculiar hemodynamic conditions.

The estimation of the size of the left ventricle seems to be useful in the differential diagnosis or evaluation of cases of septal defect associated with pulmonary hypertension. All cases with high pulmonary resistance, as a rule, show a smaller than normal left ventricular cavity. In hyperkinetic pulmonary hypertension, the left ventricular cavity is markedly or slightly enlarged. In cases with secondary pulmonary vascular occlusive disease associated with a significant increase of flow,[18] the results are variable. As described by Nadas,[18] pulmonary hypertension originates from a combination of increased resistance and increased flow ($P=R\times\mathcal{Q}$). An analysis of our series of cases reveals that the left ventricular cavity is slightly or moderately enlarged but never smaller than normal. Knowledge of the size of this cavity may be very helpful in the pathophysiologic approach in each case.

Cases of pulmonary stenosis which, in spite of severe lesions, show an end diastolic area of the left ventricle which is not smaller than normal may represent cases where a significant amount of blood passes through the patent foramen ovale, thereby increasing the amount of blood reaching the cavity of the left ventricle.

In some cases of tetralogy of Fallot, surprisingly, we have not found the small ventricle we expected. We feel that there may be several possibilities: (1) Normally the ventricular septum is oblique, forward and to the left; in tetralogy of Fallot it is transverse. It is conceivable that the thickened ventricular septum and the clockwise rotation of the heart may be capable, in some cases, of changing the shape and the area of the left ventricular cavity in frontal position. (2) Significant right to left shunt through the patent foramen ovale or through a large septal defect (pentalogy) would increase the blood flow to the left ventricle. (3) In the case of a left superior vena cava entering the left atrium, the blood reaching the left ventricle is increased. (4) Also, markedly increased bronchial circulation may cause an increase in the blood flow to the left ventricle.

The normal regression line, correlated with the B.S.A. (m.^{2}), was compared with those in each group studied. In general, cases of ventricular septal defect showed a regression line above the normal; pulmonary stenosis, atrial septal defect and tetralogy of Fallot cases showed regression lines below the normal. These findings are compatible with the pathophysiologic characteristics of each anomaly.

CORRELATION WITH HEMODYNAMIC VARIABLES

Studies were made for the correlation of the following three hemodynamic variables with the end diastolic area of the left ventricle: (1) Pulmonary flow index (P.F.I.) (L/min./m.^{2}) and the end diastolic area of the left ventricle without any correction for the B.S.A. (m.^{2}) (Fig. 6, *A–D*). (2) Pulmonary flow index (P.F.I.) (L/min./m.^{2}) and the end diastolic area of the left ventricle corrected for the B.S.A. (cm.^{2}/m.^{2}) (Fig. 7, *A–D*). (3) Pulmonary blood flow (P.B.F.) (L/min.) and the end diastolic area of the left ventricle without any correction (Fig. 8, *A–D*).

The most valuable study proved to be the correlation of the end diastolic area of the left ventricle, corrected for the B.S.A. (cm.^{2}/m.^{2}), with the pulmonary flow index (P.F.I.) (L/min./m.^{2}). A direct relationship was found in ventricular septal defect, pulmonary stenosis and tetralogy of Fallot, as expected, while cases of atrial septal defect showed an inverse relationship; *i.e.*, a small left ventricular cavity was found when a large left to right shunt was present and *vice versa*. This correlation is felt to be reliable since both parameters are reduced to a common denominator, that is, to the square meter (m.^{2}).

CONCLUSION

We believe that the estimation of the left ventricular cavity by the angiocardiographic method described is valuable and accurate. The size of the cavity is expressed as a square measure; *i.e.*, by its end diastolic area (cm.^{2}). Despite the fact that the capacity or volume of the cardiac chamber is not estimated, the enlargement or diminution of the size of this cavity can be determined precisely.

SUMMARY

1. The end diastolic area of the left ventricle in frontal angiocardiograms has been determined in 13 normal cases and the results were plotted against the body surface area (B.S.A.) (m.^{2}). Regression lines were thus obtained.

2. This area was also estimated in 75 cases of congenital heart disease. Twenty-five were cases of ventricular septal defect; 18 of atrial septal defect; 20 of pulmonary stenosis; and 12 of tetralogy of Fallot. Regression lines for each group were obtained.

3. In anteroposterior projection the normal value may be found by multiplying the body surface area (B.S.A.) (m.^{2}) by 30.1 cm.^{2}.

4. In uncomplicated ventricular septal defect, the end diastolic areas of the left ventricle were larger than normal. In the presence of high pulmonary resistance, most cases showed a left atrium of smaller size than normal. Eighty per cent of the cases of ventricular septal defect, 44 per cent of atrial septal defect, 30 per cent of pulmonary stenosis and 58 per cent of tetralogy of Fallot were found to have normal or above normal values.

5. When the normal regression line was compared with those of each group of congenital heart disease (C.H.D.), it was found that cases of ventricular septal defect showed lines above those of normal, while cases of pulmonary stenosis, atrial septal

defect and tetralogy of Fallot had below normal lines.

6. The described method for the estimation of the size of the left ventricular cavity has allowed the correlation of the pulmonary blood flow (P.B.F.) (L/min.), or the pulmonary flow index (P.F.I.) (L/min./m.^{2}) with the end diastolic area of the left ventricle, either corrected or uncorrected for the B.S.A. The differences found in cases of atrial septal defect are discussed.

7. The method is accurate and easy to perform. Single plane studies only are required. Pathophysiologic studies can be performed with correlation of different variables. This cannot be done when the size of the left ventricle is estimated by other methods.

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THE ASSESSMENT OF MITRAL VALVE LESIONS WITH CINE LEVOANGIOCARDIOGRAPHY*

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SOLITARY rheumatic valve lesions, including those of the mitral valve, present no major clinical diagnostic problem. However, the determination of the severity and clinical significance of a lesion in the presence of multiple murmurs and/or presumed multiple valve involvement can be difficult.

Several years prior to the advent of valve replacement, the patient suspected of having other than solitary mitral stenosis was usually studied with catheterization alone. This approach was terminated by our experience with 2 patients who were erroneously submitted to surgery for correction of mitral stenosis by closed commissurotomy. Both had marked mitral insufficiency and 1 died at surgery. Since then cardiac catheterization and angiography were undertaken routinely in patients with suspected multiple lesions so as to exclude the patients who would not be helped by mitral commissurotomy. With the development of valve prosthetic devices, these techniques have been employed to aid in the selection of the type of surgical procedure to be employed. In view of the fact that in most institutions an appreciable difference exists in the surgical mortality of open and closed heart procedures and between single and multiple valve replacement operations, these procedures have more than an academic significance.

The accuracy of levoangiocardiography in the assessment of the severity of valvular lesions of the left heart has been reported by several authors.[2,3,4,6,7] In all of these studies, a rapid film changer was used. Because of the temporary instability of our Schönander film changer, we began and still employ a cine technique with a 9 inch (22 cm.) image amplifier (Picker amplifilmer).

TECHNIQUE

The angiocardiography has routinely been performed in conjunction with catheterization and recording of pressure studies. A detailed description of the pressure curve analysis will be published elsewhere.

Injections were made into the left atrium via a trans-septal catheter, into the left ventricle via a catheter passed retrograde across the aortic valve and, if there was any clinical suggestion of aortic valve disease, into the ascending aorta. Twenty to 40 cc. of ditriokon (diprotrizoate and diatrizoate mixture) was injected according to the estimated volume of the chamber injected. Using a Taveras injector, a pressure of 275 pounds per square inch through a No. 8 French 70 cm. catheter was employed for the left atrial injections and 625 pounds per square inch through a No. 6 N.I.H. catheter for the ventricular and ascending aortic injections. Filming was almost uniformly accomplished with 30 frames per second, being reduced to 15 frames per second in patients with larger hearts. For ventricular and atrial injections, left lateral and/or right anterior oblique projection studies were made. For ascending aortic injections, left lateral studies were obtained.

METHODS OF ANALYSIS

A retrospective analysis of 67 patients who had had a complete catheterization with a cine study and examination of the mitral valves at surgery was made.

The surgical estimates of the severity of the lesions were routinely performed by one or more of the 3 attending cardiac surgeons at this institution. Stenotic lesions were estimated by palpation and/or inspection. The estimates were recorded in square cen-

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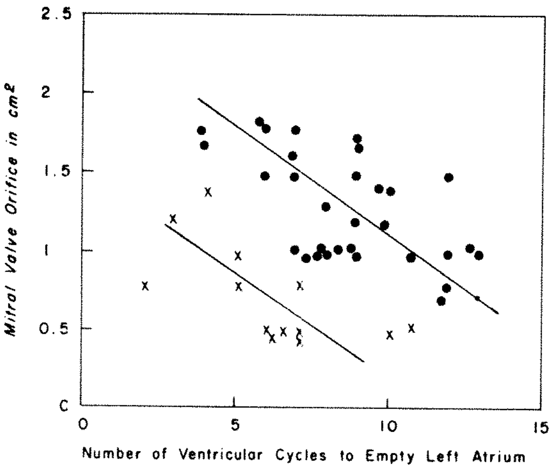

FIG. 1. Mitral stenosis. Emptying time of the left atrium expressed in ventricular cycles versus surgical estimate of the size of mitral valve orifice. Closed circles indicate cases with auricular fibrillation. X's represent individuals with normal sinus rhythm.

timeters from prior planigraphic measurements of various levels on their examining fingers. Insufficiency was estimated by palpation of the jet and expressed either in cubic centimeters per beat or by classification into 4 grades: absent, mild, moderate, and severe.

The cine angiocardiographic estimates were made independently of the catheterization data. The lesions of stenosis and insufficiency of the mitral valve were

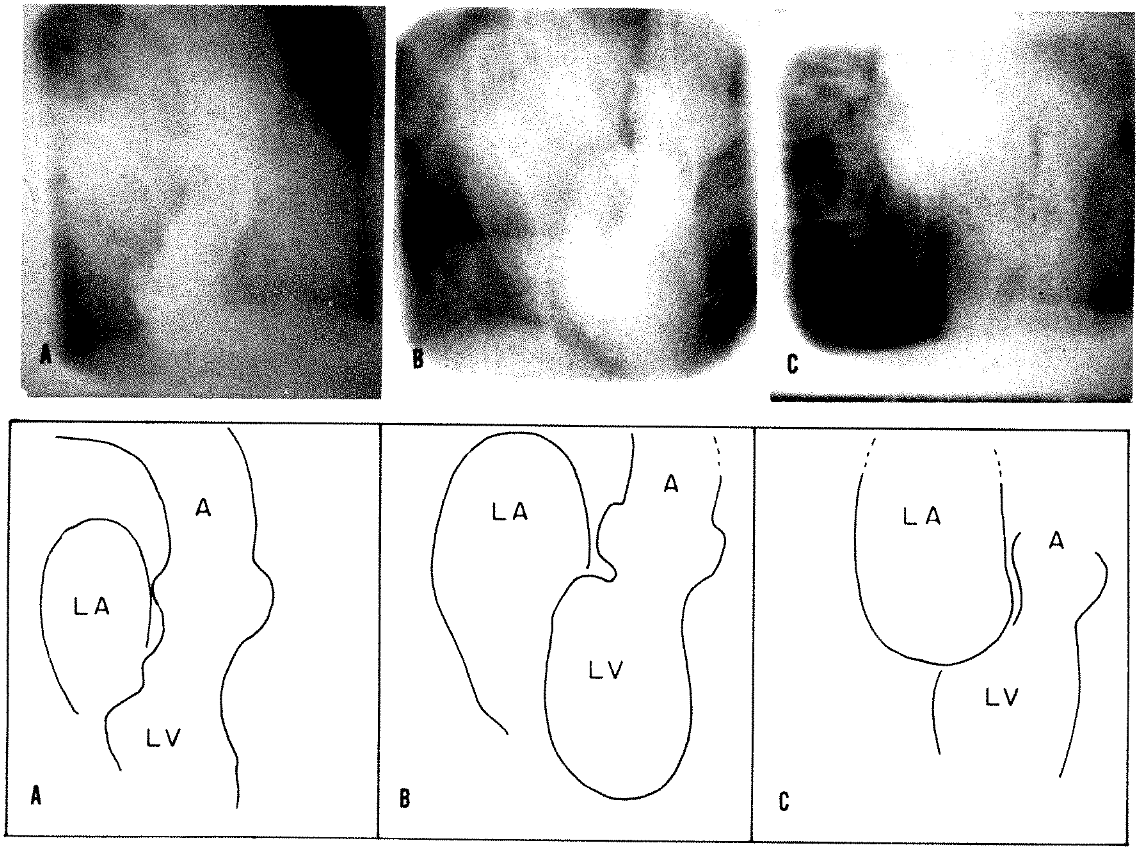

FIG. 2. Individual cine frames following left ventricular injection. (*A*) Mild mitral insufficiency. Faint opacification of minimally enlarged left atrium. Note the head of the contrast medium beyond the aortic arch. (*B*) Moderate insufficiency. Opacification of a large left atrium. The leading edge of the contrast material is beyond the ascending aorta. (*C*) Marked insufficiency. Marked opacification of massive left atrium. The leading edge of the contrast medium is at the level of the sinuses of Valsalva. LA = left atrium; A = aorta; LV = left ventricle.

graded as absent, mild, moderate or marked.

Using the published angiographic criteria for the evaluation of mitral valve lesions, *i.e.*, those possible with cine studies, the following factors were analyzed and graded 0 to 3 (absent, mild, moderate or marked):

1. Size of the left atrium.
2. Size of valve jet (prograde and retrograde).
3. Number of ventricular cycles to empty the left atrium after the end of atrial injection.
4. Degree of opacity of left atrium following ventricular injection.
5. Number of ventricular cycles to reach maximum left atrial opacity after ventricular injection.
6. Position of the leading edge of the bolus in the aorta (valve, ascending, arch, descending) at the time of complete opacification of the left atrium.
7. Size of the left ventricle.

This general evaluation and the specific signs were correlated with the surgical data.

RESULTS

The left atrial size correlated generally with the degree of mitral valvular disease, being "moderately" and "markedly" enlarged in severe combined lesions and lesions with predominant insufficiency. However, it was occasionally only "mildly" enlarged in cases with severe, relatively pure stenotic lesions.

The size of the regurgitant jet correlated generally with the degree of insufficiency judged at surgery. A distinct prograde jet was visualized only in one-third of the cases of mitral stenosis.

The number of ventricular cycles required to *empty the left atrium* seemed to correlate with the degree of mitral stenosis. In cases of predominant and relatively pure stenotic lesions, there was good correlation. The presence of atrial fibrillation versus normal sinus rhythm appeared to have an effect on this phenomenon (Fig. 1).

The extent and *degree of left atrial opacification* following left ventricular injection generally correlated well with the degree of regurgitation. The number of *ventricular cycles to reach maximum left atrial opacity alone* is of limited value and rhythm dependent (Fig. 2, *A–C*).

The *position of the leading edge of the contrast agent* in the thoracic aorta at the time the left atrium is completely opacified (following left ventricular injection) correlated well with the degree of regurgitation recorded at surgery. Because of the size of the field, this sign could be assessed in only about one-half of the cases.

The *size of the left ventricle* generally correlated with the degree of mitral insufficiency.

DISCUSSION

MITRAL STENOSIS

The evaluation of the severity of mitral stenosis as an isolated or predominant lesion can be accomplished by levocineangiography. Visualization of a jet is an inconsistent phenomenon which may be due to the difficulty of obtaining a "profile" view of the mitral valve[5] and the size of the left atrium seems to bear little relationship to the severity of the stenosis. However, the number of ventricular cycles necessary to empty the atrium is a consistent correlating factor and must be judged in respect to the atrial rhythm. In cases of significant mitral insufficiency, the severity of a stenotic lesion cannot be judged by injection of the left atrium alone.

Left atrial-ventricular diastolic pressure gradients before and, particularly, after levoangiocardiography are probably the methods of choice in evaluating the severity of mitral stenosis, particularly, in cases with significant mitral insufficiency.

MITRAL INSUFFICIENCY

Mitral insufficiency is conveniently and accurately evaluated by left ventricular injection in cases with relatively pure or mixed lesions. As previously described,[2,4,7] an accurate method is by the quantitation of the opacity in the left atrium relative to its size. A moderate sized regurgitant jet may be severely diluted in a massive enlarged left atrium and thus underestimated unless this quantitation is performed. The position of the leading edge of the bolus in the aorta (as originally described by Björk *et al.*[2]) was also found to be an accurate indicator of severity. The volumetric assessment of chamber size as proposed by Arvidsson[1] seems unnecessary for clinical assessment.

As our experience has increased, the clinical importance of valvular calcification has become more apparent, and studies of the mitral valve lesions are now rarely performed in individuals with extensive mitral valvular calcification. Because of previous experience, we feel that these patients do poorly with closed commissurotomy and they are operated on with the open heart technique regardless of the relative degree of stenosis and insufficiency.

CONCLUSIONS

Levoangiocardiography can aid in the assessment of the severity of mitral stenosis in cases of pure and relatively pure lesions. Injection into the left atrium and left ventricle is necessary to assess the severity of stenosis in a mixed stenotic and insufficiency lesion of the mitral valve. The severity of mitral stenosis cannot be evaluated simply and the rhythm of the heart must be considered in all cases. The diastolic gradient across the valve after angiography (or some similar stress) is thought to be more accurate.

Levoangiocardiography with left ventricular injection alone is a simple and accurate technique for evaluating mitral insufficiency in a pure or mixed state. The degree of left atrium opacification correlated with the size of the left atrium is the most accurate indicator. Where it could be observed, the position of the leading edge of the bolus in the aorta at the time of complete left atrial opacification seemed equally accurate. This angiographic technique is probably better and certainly more direct than pressure curve analysis.

The cine technique seems a reasonable alternative to the classic simultaneous biplane large film study described in the majority of the articles on the subject, since chamber volume assessment seems unnecessary. In cases of isolated mitral valvular disease with extensive calcification, neither catheterization nor angiographic studies seem clinically indicated prior to open heart surgery.

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NEW CONCEPTS IN DEXTROCARDIA*

ANGIOCARDIOGRAPHIC CONSIDERATIONS

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DEXTROCARDIA, a positional anomaly in which the heart is predominantly in the right hemithorax, has been a fascinating curiosity since ancient times. Advent of the stethoscope and more particularly of the roentgenographic examination of the chest facilitated ante mortem detection of dextrocardia and stimulated the search for better understanding of the anomaly. It was soon appreciated that several types of dextrocardia existed and that some were regularly associated with other malformations of the heart which resulted in serious cardiac disability.

Three to five per cent of individuals with congenital heart disease have dextrocardia. With accurate diagnosis surgical correction of some of the associated anomalies in these patients is now possible. The radiologist has a special position in this problem of dextrocardia, not only because of the original roentgenographic demonstration of the anomaly, but also because precise preoperative delineation of cardiac anatomy in these complex cases can only be done by angiocardiography.

This report is concerned with primary congenital dextrocardia, not with dextrocardia secondary to congenital extracardiac factors (as chest wall anomalies, lung hypoplasia, hernias, etc.) nor with acquired dextrocardia (due to lung resection, atelectasis, etc.). Our study is based upon 59 cases of dextrocardia seen during the past 9 years. The ages ranged from 1 day to 68 years. All had chest roentgenograms. Angiocardiographic examination was performed in 40 cases; 13 of these and 8 others were examined at necropsy. The remaining 11 cases, including 6 with situs inversus, have not yet been studied by angiocardiography.

ANATOMIC AND DEVELOPMENTAL CONSIDERATIONS

Identification of visceral and atrial situs (or position) development and recognition of two types of bulboventricular loop development are essential to understanding *primary congenital dextrocardia.* The sometimes associated anomaly, transposition of the great vessels, will be discussed in a later section.

A. SITUS OF VISCERA AND ATRIA

Determination of viscero-atrial situs is the first step in analysis of dextrocardia. Situs solitus refers to usual or normal viscero-atrial relationships. Situs inversus, on the other hand, is the inverse or apparent "mirror-image" of situs solitus (inversion here means opposite or "mirror-image" development of right and left relationships with preservation of ventrodorsal relationships). Indeterminate or mixed situs development may also occur—often with symmetric instead of the normal asymmetric liver formation and sometimes without a spleen (asplenia).

Chest roentgenograms establish the diagnosis of dextrocardia. The gastric air bubble and the liver shadow indicate with considerable certainty whether situs solitus, situs inversus or indeterminate visceral situs is present.

The situs of the great veins and viscera, particularly of the liver, indicates the atrial situs with great accuracy.[35] In normal development, situs solitus, the right atrium is on the right; in situs inversus, as might be predicted, the anatomic right atrium is on the left. Since the atria are attached to the great veins, they are relatively fixed in a position dorsal to the ventricles.

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B. BULBOVENTRICULAR LOOP FORMATION

The human fetal heart develops from a primitive tubular structure formed by fusion of two adjacent endothelial tubes. The still paired sinus venosus, the atrium, the ventricle, the bulbus and the truncus are connected in series as shown diagrammatically in Figure 1*A*. This tubular heart bends as it loses its dorsal mesocardium in the bulboventricular region and also lengthens more rapidly than the pericardial cavity. There are two major possibilities, *i.e.*, a rightward or a leftward bending of the cardiac tube. In normal heart development, this bending of the bulboventricular region is toward the right (concordant D–LOOP). Alternately and rarely, the bulboventricular loop bends oppositely to the left (discordant L–LOOP). Concordance here refers to normal or expected bulboventricular

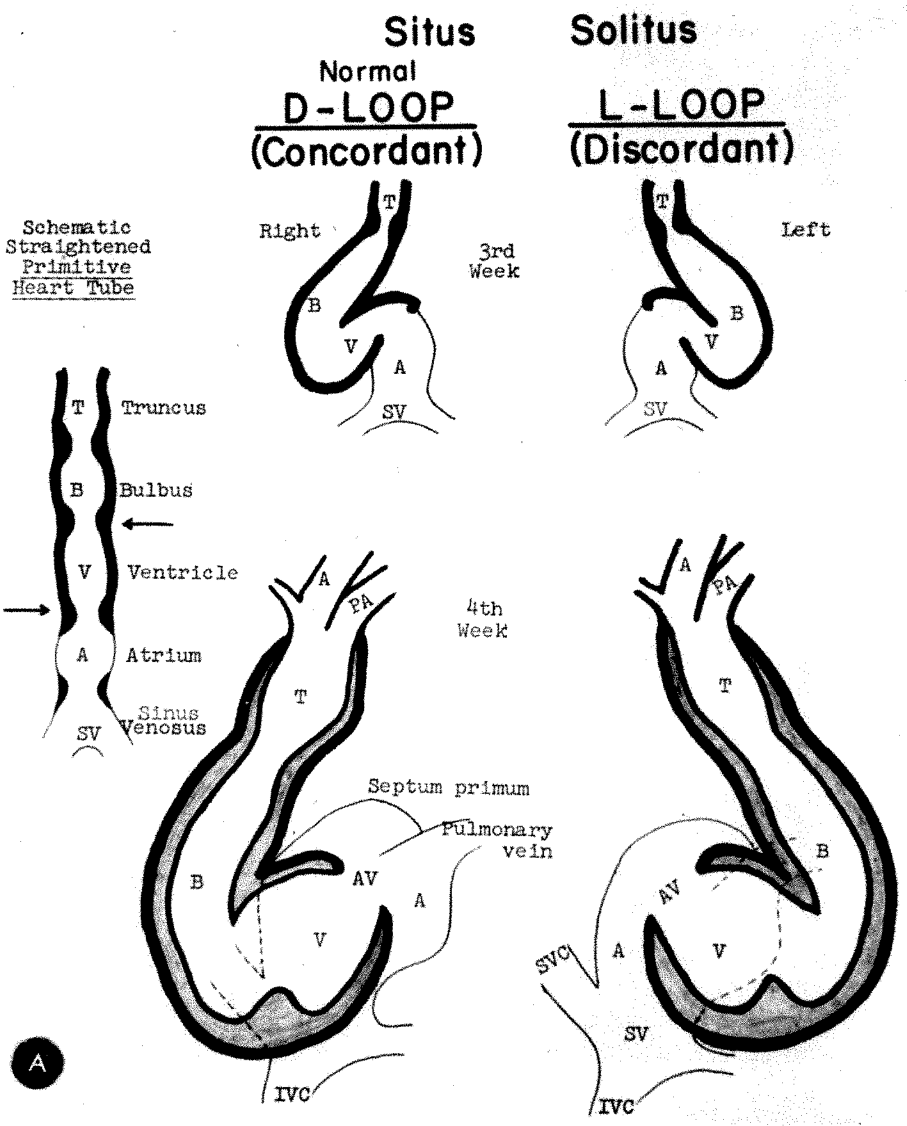


Fig. 1. Diagrams of cardiac development in situs solitus. (*A*) On the left is a diagram of primitive tubular heart with truncus, bulbus, ventricle and atrium in series. In the middle are 2 diagrams showing the normal (concordant) bending of the bulboventricular loop to right (D-LOOP). On the right 2 diagrams show abnormal (discordant) bowing of the bulboventricular loop to left (L-LOOP).

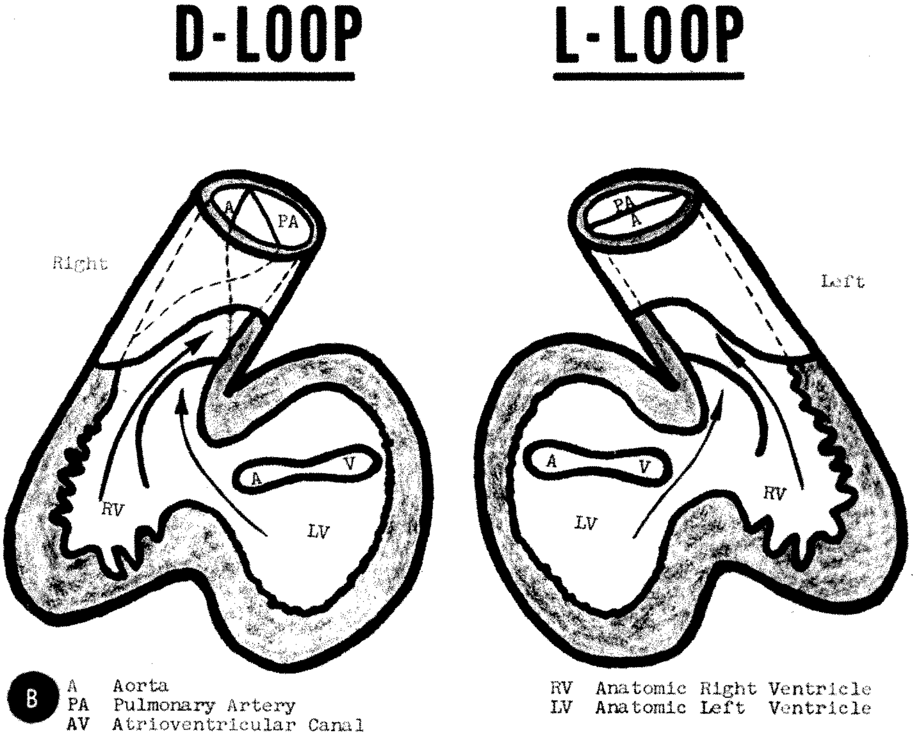

D-LOOP
L-LOOP
Right
A
PA
RV
LV
A
V
Left
PA
A
RV
LV
A
V
B
A Aorta
PA Pulmonary Artery
AV Atrioventricular Canal
RV Anatomic Right Ventricle
LV Anatomic Left Ventricle

Fig. 1. (*B*) Frontal hemisections of bulboventricular loop. Note in D-LOOP development the right ventricle is on right and the left ventricle is on left. In L-LOOP development the ventricular positions are inverted—and characteristically transposition of the great vessels is present, *i.e.*, the aorta arises from the anatomic right ventricle.

bending or loop formation with respect to viscero-atrial situs: *i.e.*, a D-LOOP in situs solitus or an L-LOOP in situs inversus; conversely an inappropriate L-LOOP in situs solitus or a D-LOOP in situs inversus is regarded as discordant. Discordant bowing of the bulboventricular loop, *e.g.*, a discordant L-LOOP in situs solitus, is thought to be the fundamental defect (bulboventricular inversion) in the cardiac anomaly referred to as "congenitally corrected transposition of the great vessels."

De la Cruz *et al.*[8] and recently Van Praagh *et al.*[36] have brilliantly discussed dextrocardia and the latter have designated rightward bending of the bulboventricular loop as a D-LOOP and leftward bending as an L-LOOP. In Figure 1, *A*, *B* and *C* diagrams in frontal projection show the consequences of normal concordant D-LOOP and of discordant L-LOOP formation in situs solitus. Certain features of cardiac development which pertain to the position of the ventricles and great vessels should be particularly noted:

1. The anatomic right ventricle develops from the bulbus, and the anatomic left ventricle from the primitive ventricle. As can be seen from the diagrams in Figure 1, *A*, *B* and *C* the anatomic right ventricle (bulbus) is positioned on the right of the anatomic left ventricle when the bulboventricular loop bends to the right, D-LOOP (normal or concordant in situs solitus but discordant in situs inversus).

Conversely, when the bulboventricular loop bends to the left, the anatomic right ventricle is positioned to the left of the anatomic left ventricle, L-LOOP, (discordant in situs solitus but concordant in situs inversus).

2. When bulboventricular loop develop-

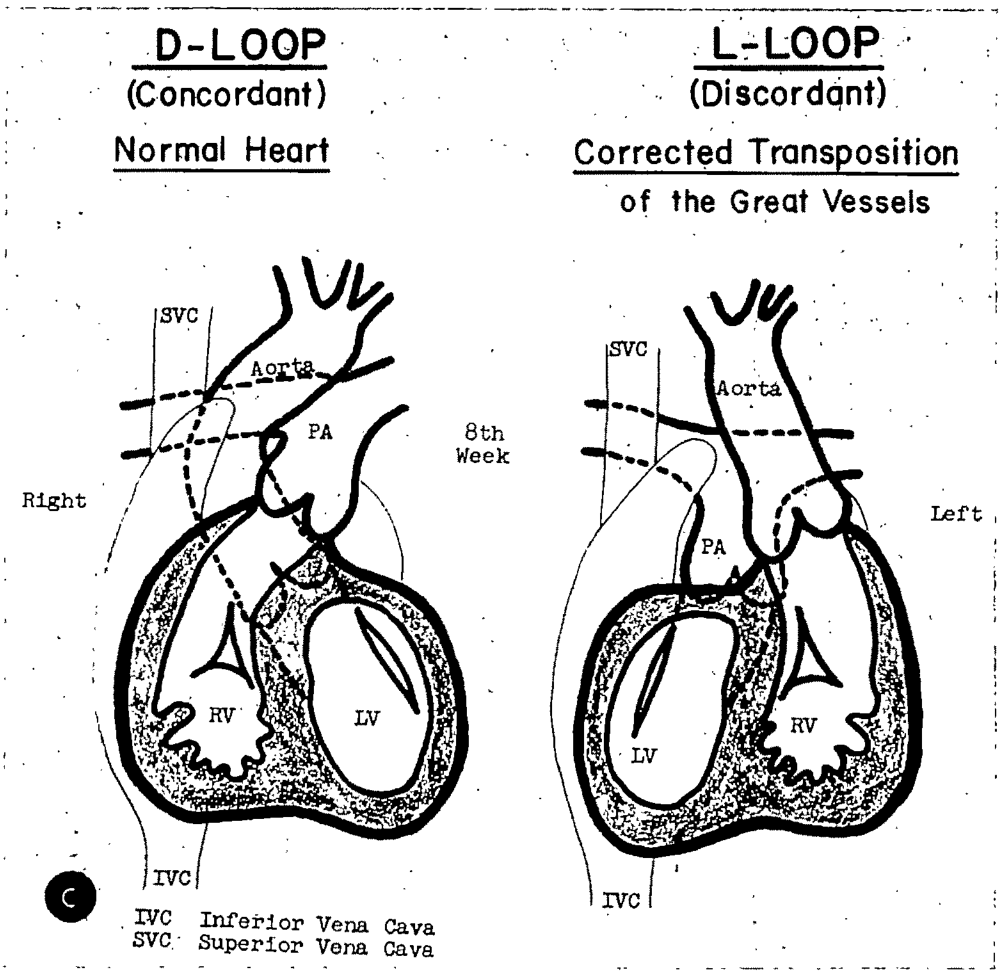


Fig. 1. (*C*) The concordant D-LOOP development when completed results in the normal left sided heart in situs solitus. Discordant L-LOOP development, if complete, results in the anomaly known as "corrected transposition of the great vessels." Quite often the ventricles produced by an L-LOOP will shift to right to produce a type of dextrocardia. Cardiac loop development is usually completed by the end of second month of fetal life.

ment is discordant to viscero-atrial situs (L-LOOP in situs solitus or D-LOOP in situs inversus), transposition of the great vessels is regularly present. This type of transposition is potentially congenitally corrected because the atrioventricular connections are also reversed (the anatomic right atrium is connected to the anatomic left ventricle, but, because of the great vessel transposition, this anatomic left ventricle is connected in turn to the pulmonary artery. Similarly, the anatomic left atrium is connected to the anatomic right ventricle and this to the transposed aorta).

3. The truncus arteriosus has a relatively cephalad and ventrad position in the embryo. Thus the outflow valve (semilunar valve) connecting the anatomic right ventricle (bulbus) to the truncus is characteristically cephalad and ventrad in position—regardless of the type of situs or bulboventricular loop—or of the presence or absence of transposition of the great vessels. The other semilunar valve which is transferred to the anatomic left ventricle (primitive ventricle) is necessarily dorsad and caudad to the semilunar valve of the anatomic right ventricle.

4. Although the bulboventricular loop initially bends to the right (D-LOOP in

situs solitus) the ventricular region of the heart later shifts to the left to form the normal left-sided heart. Conversely, despite initial bending of the bulboventricular loop to the left in situs inversus (concordant L–LOOP) the ventricular region of the heart shifts later to the right to produce "mirror-image" dextrocardia. Thus, a concordant D–LOOP characteristically produces a left-sided heart and a concordant L–LOOP typically produces a right-sided heart.

In contrast, when bulboventricular loop formation is discordant or inappropriate to viscero-atrial development, the later shift of the ventricular region to the side opposite that of initial bowing of the bulboventricular loop tends to occur but usually does not actually take place. Extrinsic factors probably interfere.

MECHANISMS IN THE PRODUCTION OF DEXTROCARDIA

Consideration of the viscero-atrial situs together with the direction of bulboventricular loop bending suggests several mechanisms that are involved in the production of dextrocardia (Fig. 2, *A–E*).

TYPE IA—DEXTROCARDIA IN SITUS SOLITUS WITH CONCORDANT D-LOOP

In normal development, the bulboventricular loop bends initially to the right, placing the ventricular region and particularly the future right ventricle (bulbus) on the right (situs solitus with concordant D–LOOP). During the second month of fetal life, the ventricular region normally shifts toward the left and rotates clockwise to produce the normal left-sided heart and to place the right ventricular outflow tract on or close to the left heart border.

Failure of this final leftward shift of the ventricles is the basis for Type IA dextrocardia (Fig. 2*A*).

TYPE IB—DEXTROCARDIA IN SITUS SOLITUS WITH DISCORDANT L-LOOP

Conversely, when the discordant bulboventricular loop bends to the left, it places the bulboventricular region and particularly the future anatomic right ventricle on the left (situs solitus with discordant L–LOOP). Both ventricles tend later to swing to the right, mimicking inversely the normal ventricular development.

A marked rightward shift of the discordant ventricles produces Type IB dextrocardia (Fig. 2*B*).

TYPE IIA—DEXTROCARDIA IN SITUS INVERSUS WITH CONCORDANT L-LOOP
(mirror-image dextrocardia)

In situs inversus, "mirror-image" development of the normal usually occurs with the bulboventricular loop bending initially to the left placing the ventricles mainly on the left (situs inversus with concordant L–LOOP). Subsequently, the ventricular region swings to the right producing the "mirror-image" of the normal heart.

This normal development in situs inversus results in Type IIA dextrocardia (Fig. 2*C*).

TYPE IIB—DEXTROCARDIA IN SITUS INVERSUS WITH DISCORDANT D-LOOP

In situs inversus, a discordant bulboventricular loop bends initially to the right, placing the ventricular region and particularly the future anatomic right ventricle (bulbus) mainly on the right (situs inversus with discordant D–LOOP). The ventricular region later tends to swing toward the left.

Failure of this final leftward shift of the discordant ventricles is the basis for Type IIB dextrocardia (Fig. 2*D*).

TYPE III—DEXTROCARDIA IN INDETERMINATE SITUS

When viscero-atrial situs development is not dominated by either right-sided or left-sided structures but rather is abnormally symmetric, situs development is regarded as indeterminate. Concordance and discordance in bulboventricular loop formation is no longer meaningful though D, L or indeterminate loops may be present.

Dextrocardia in indeterminate situs is the Type III dextrocardia (Fig. 2*E*).

Rarely, bulboventricular loop development also is indeterminate, *i.e.*, neither typical D–LOOP nor typical L–LOOP de-

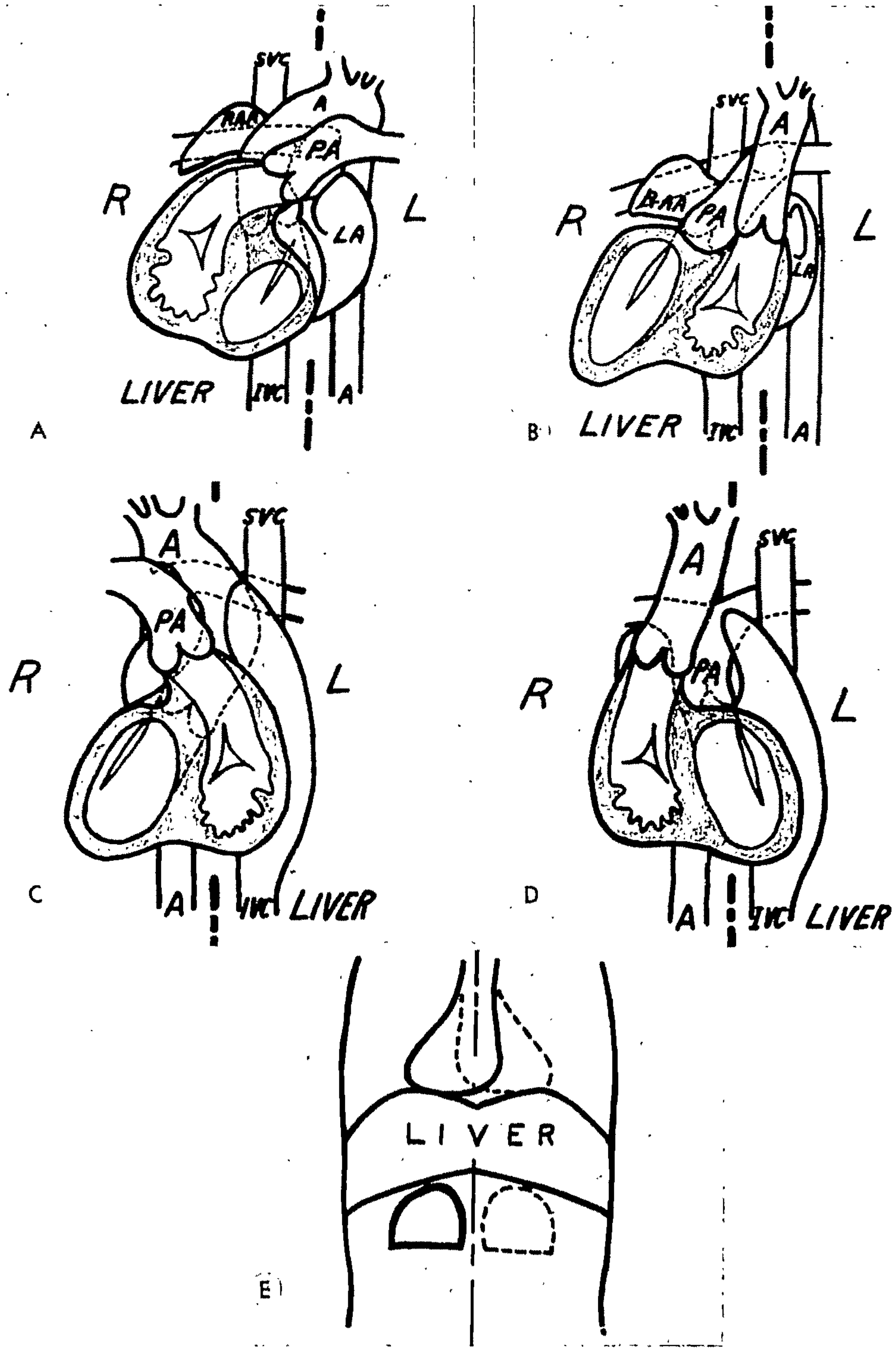


Fig. 2.
(*A*) Type IA. Dextrocardia in situs solitus with concordant D-LOOP.
(*B*) Type IB. Dextrocardia in situs solitus with discordant L-LOOP.
(*C*) Type IIA. Dextrocardia in situs inversus with concordant L-LOOP.
(*D*) Type IIB. Dextrocardia in situs inversus with discordant D-LOOP.
(*E*) Type III. Dextrocardia in indeterminate situs.

velopment occurs, but rather the loop develops in some other way, most often by bowing dorsally.[36] Though extremely rare, and then usually associated with a common ventricle, this category is included for completeness in our classification of dextrocardia (Types IC, IIC and IIIC).

On the basis of these variations in viscero-atrial situs and in bulboventricular loop development a classification of dextrocardia is suggested:

CLASSIFICATION OF DEXTROCARDIA

I *Situs Solitus*
  A. *Concordant—D-bulboventricular loop*
  B. *Discordant—L-bulboventricular loop*
  C. Indeterminate bulboventricular loop

II *Situs Inversus*
  A. *Concordant—L-bulboventricular loop* (mirror-image dextrocardia)
  B. *Discordant—D-bulboventricular loop*
  C. Indeterminate bulboventricular loop

III *Indeterminate Situs* (includes asplenia)
  A. D-bulboventricular loop
  B. L-bulboventricular loop
  C. Indeterminate bulboventricular loop

ANGIOCARDIOGRAPHIC DEMONSTRATION OF THE HEART CHAMBERS

Angiocardiography is essential to the delineation of bulboventricular loop development: cardiac chambers must be identified and the great vessels must be demonstrated.

The positions of the atria, most reliably indicated by viscero-atrial situs on roentgen and angiocardiographic examination, are also usefully reflected on the electrocardiogram: The P wave in lead 1 is usually positive when the anatomic right atrium is on the right and negative when the anatomic right atrium is on the left, *e.g.*, situs inversus.

Lev and Rowlatt[16,17] have pointed out that "heart chambers cannot be adequately named according to the type of blood they carry or according to their proximal or distal connections. Chambers are best named according to morphology and (in gross pathology) the morphology is best recognized by observing the architecture of the septum." Preoperative clinical identification of cardiac chambers cannot, however, be done by direct inspection and depends primarily upon angiocardiography. Listed below are angiocardiographic features of value in identification of the atria and ventricles. The double starred features are of good reliability, the single starred features of some value and the other items of questionable value.

Right Atrium (limbus and fossa ovalis on septal surface)
  **1. Visceral situs: on side of liver (usually opposite gastric air bubble)
  **2. Receives inferior vena cava
  *3. Receives coronary sinus
  *4. Vertical long axis
  5. Receives superior vena cava

Left Atrium (irregular surface of septum primum)
  **1. Visceral situs: opposite liver
  *2. Receives pulmonary veins
  *3. Transverse long axis–oval shape
  4. Relatively cephalad position

Right Ventricle (sinus separated from infundibulum by crista, posterior septum trabeculated with papillary muscle, tricuspid atrioventricular valve)
  **1. An infundibulum separates atrioventricular valve from semilunar valve (*i.e.*, inlet from outlet)
  *2. Semilunar valve more ventrad and cephalad
  *3. Coarse trabeculation—especially on septal surface

Left Ventricle (no infundibulum, smooth septal surface, bicuspid atrioventricular valve)
  **1. No infundibulum
  *2. Semilunar valve more dorsad and caudad
  3. Fine trabeculation

Sometimes, no separation between the atria or between the ventricles exists so that either a *common atrium* or *common ventricle* is present. A common atrium is often recognized by angiocardiography, and, in any case, does not offer an impossible situation to the cardiac surgeon. A common ventricle, however, is a more serious problem since it is not usually considered to be surgically correctable. It is found in 10–20 per cent of cases of dextrocardia[36] and in 50 per cent of

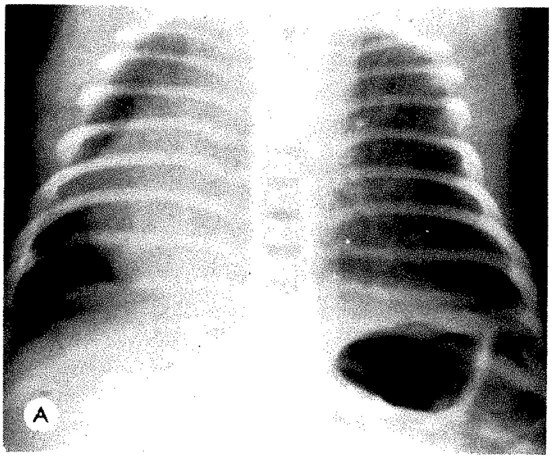
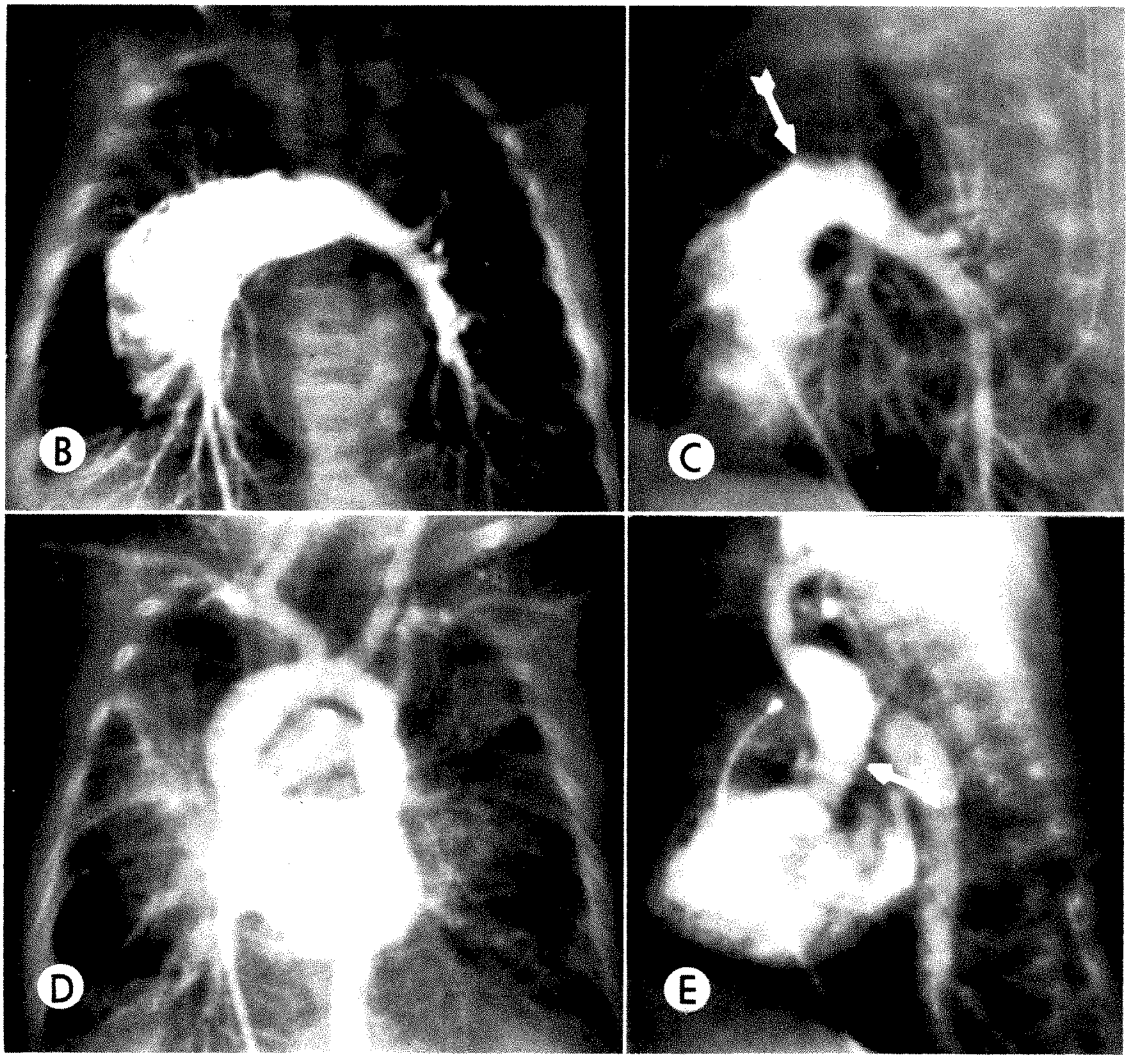


Fig. 3. Type IA. Dextrocardia in situs solitus with concordant D-LOOP. (*A*) Chest roentgenogram showing dextrocardia in situs solitus.

those with asplenia or indeterminate situs.[25]

The angiocardiographic diagnosis of common ventricle may be difficult. The subject has recently been intensively studied,[3,34,37] though there is not yet complete agreement on its definition, or on the designation "single" as opposed to "common" ventricle. By usual definition, a common ventricle receives both the tricuspid and mitral valves or a common valve. Therefore, a common ventricle may be excluded if selective injections of contrast material into the right atrium and left atrium, respectively (or into the pulmonary artery if the left atrium cannot be entered), demonstrate that the atria empty into different ven-



tricles. In many instances selective ventricular injections are also helpful[37] and sometimes an aortogram demonstrating coronary artery anatomy may be useful.[25]

In most cases of common ventricle (80 per cent) the anatomic right ventricular sinus or inflow tract is said to be absent.[34] Then, a large anatomic left ventricle with its usual outflow tract is present together with a ventricular septal defect leading into a secondary rudimentary outflow tract or infundibulum (representing the right ventricular infundibulum). In the great majority of cases (85 per cent), the great vessels are transposed so that the aorta arises from the rudimentary outflow tract, which may be either on the right or left side of the base of the heart, possibly but not necessarily corresponding to D or L bulboventricular loop development respectively.

TYPE IA DEXTROCARDIA IN SITUS SOLITUS WITH CONCORDANT D-LOOP

This is the most common type of dextrocardia in our experience. Of the 26 cases observed in this group, 17 had serious associated intracardiac defects; 4 of these had transposition of the great vessels—an anomaly which only occasionally occurs in concordant bulboventricular loops. A total of 9 (9/26) adults was in this group; 4 of whom had no apparent cardiac anomalies. Five of 17 children had no associated intracardiac anomalies. Angiocardiograms (Fig. 3, *B–E;* and 4, *B–E*) showing 2 of these cases are presented. Five patients with no clinical evidence of heart disease have not yet had angiocardiographic study, but are thought to be examples of Type IA instead of IB, mainly because of the rarity of normally functioning hearts with discordant loop development.

In this form of dextrocardia, the relationships of the cardiac chambers relative to each other and to the great vessels are basically normal, whether the dextrocardia is the result of primary or secondary congenital factors or is acquired after birth. In all these cases, situs solitus with concordant D-LOOP development is present. Since this situs and type of bulboventricular loop are also found in the normal left-sided heart, nearly all cases of secondary dextrocardia, particularly those acquired after birth, are of this type also.

Right lung hypoplasia occurred in 12 additional individuals, not included in the above figures because of the possibility that the dextrocardia was secondary to the lung anomaly. All had dextrocardia in situs solitus with concordant D-LOOP. Pulmonary hypoplasia was not obvious in our other types of dextrocardia. Such right lung hypoplasia might interfere with normal shift of the cardiac ventricles into the left chest. On the other hand, we consider it as likely that primary dextrocardia or its cause may interfere with right lung development, the cardiac anomaly then being primary. These patients with hypoplasia of the right lung typically had small pulmonary arteries, often had no eparterial bronchus, and half had anomalous venous drainage of the right lung into the right atrium

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FIG. 3. (*B* and *C*) Simultaneous frontal and lateral angiocardiograms showing selective opacification of the right ventricle and pulmonary arteries. Note the rightward position of the right ventricle and ventrad position of the pulmonary valve (arrow). At 2 months of age, severe dyspnea was intermittently present and often seemed dependent upon position. No intracardiac anomalies were present.

(*D* and *E*) Two seconds later left heart is opacified together with a mild coarctation of the aorta and faint reopacification of the pulmonary artery due to patent ductus arteriosus. Repair of coarctation and patent ductus did not alter progressive respiratory difficulties. At necropsy, no specific cause of death was found. The extreme dorsad position of the ascending aorta (arrow) and aortic arch may have compressed the trachea, the right bronchus, and the esophagus. This type of compression was considered significant in the death of Case 4 of Van Praagh *et al.*[35]

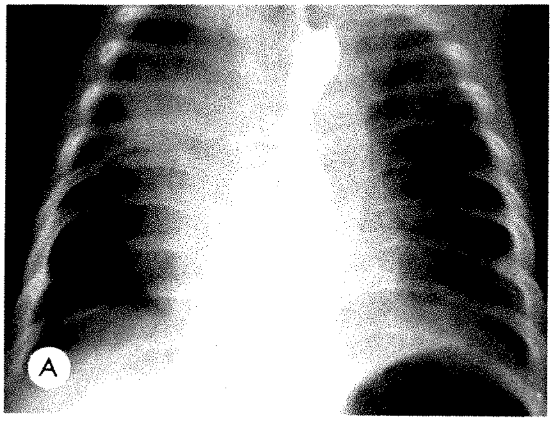
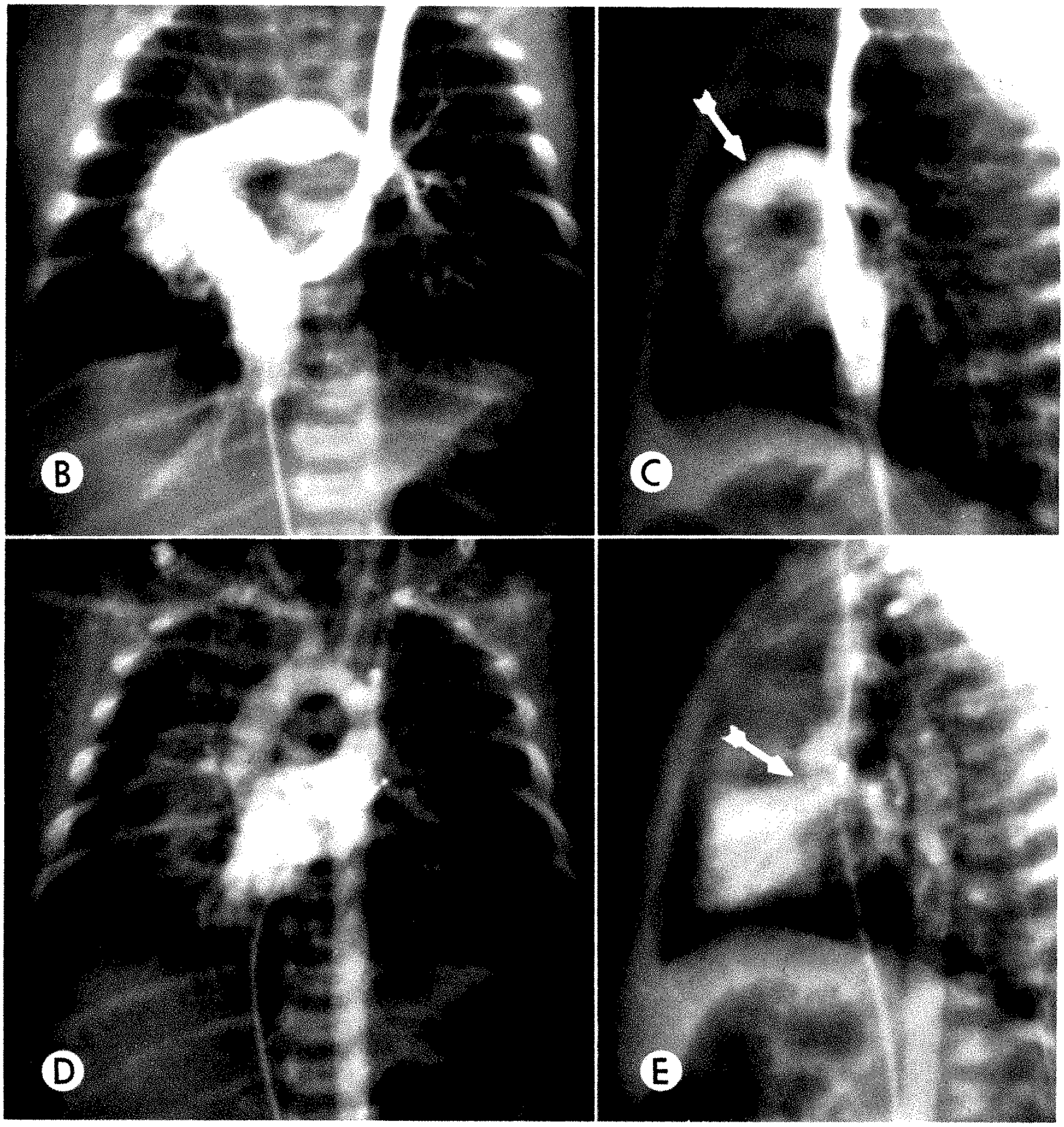


Fig. 4. Type IA. Dextrocardia in situs solitus with concordant D-LOOP. (*A*) Chest roentgenogram showing dextrocardia in situs solitus. Barium is in esophagus.

or inferior vena cava (Fig. 5, *A* and *B*). Separation of these cases from Type IA dextrocardia is arbitrary since the basic cardiac structure is the same (situs solitus with concordant D-LOOP), and it may be equivocal whether or not lung hypoplasia is present.



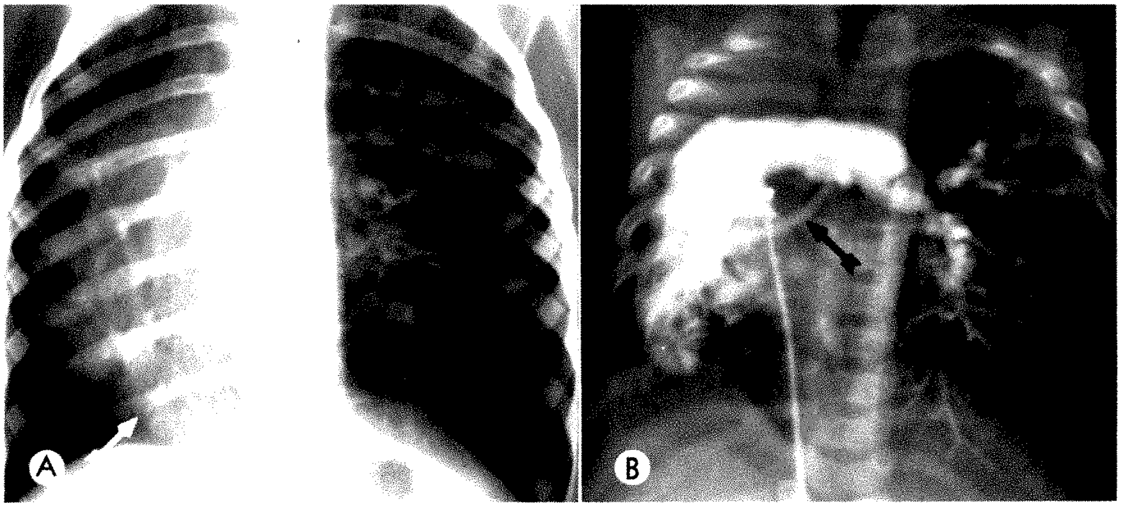


Fig. 5. Secondary congenital dextrocardia in situs solitus with concordant D-LOOP. (*A*) Frontal chest roentgenogram showing heart shifted to right and anomalous vascular shadow in right retrocardiac region (arrow). This represents an anomalous pulmonary vein entering inferior vena cava (scimitar syndrome). The dextrocardia was considered secondary to hypoplastic right lung.

(*B*) In another case a frontal angiocardiogram demonstrates opacified right heart shifted to right secondary to hypoplastic right lung. Note small right pulmonary artery (arrow). No intracardiac defects are present; opacification of the descending aorta is via a patent ductus arteriosus. Often it is arbitrary whether the dextrocardia is regarded as primary or secondary.

TYPE IB DEXTROCARDIA IN SITUS SOLITUS WITH DISCORDANT L-LOOP

We have encountered 10 cases in this group. Two of these were adults: 1 a 34 year old man with a large ventricular septal defect and the other a 30 year old man with no other defects except for complete heart block. All of the 8 children had intracardiac septal defects except for 1 asymptomatic child with Ebstein's anomaly of the left atrioventricular valve whose angiocardiograms (Fig. 6, *B*–*E*) afford excellent demonstration of Type IB dextrocardia.

All of these cases had transposition of the great vessels in that the aorta arose from the anatomic right ventricle. Transposition is nearly always present when bulboventricular loop formation is discordant relative to viscero-atrial situs. This combination of anomalies is the basis for "congenitally corrected transposition of the great vessels" as discussed above under "bulboventricular loop formation." Dextrocardia is reported in 5–15 per cent of cases with "corrected transposition"[8,11,26] while another 5–15 per cent of these patients have mesocardia—a midline heart or "partial" dextrocardia.

Cardiac septation is seldom normal when bulboventricular loop development is discordant to viscero-atrial situs. Indeed, serious intracardiac anomalies are the rule, being found in more than 90 per cent of cases. Among infants who succumb to congenital heart disease, this is apparently the

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Fig. 4. (*B* and *C*) Simultaneous frontal and lateral angiocardiograms after injection of contrast material into an anomalous left superior vena cava which is connected to the right atrium via the coronary sinus; no other anomalies were found in this asymptomatic baby. The right heart is on the right and the pulmonary valve (arrow) is ventrally located at outlet of the anatomic right ventricle.

(*D* and *E*) When the left heart is opacified 3 seconds later, the aorta is shown to arise from the anatomic left ventricle. As expected, the aortic valve (arrow) is caudad, dorsad and to the right of the pulmonary valve. Intrinsically normal relationships are present between the ventricles and great vessels in this type of dextrocardia.

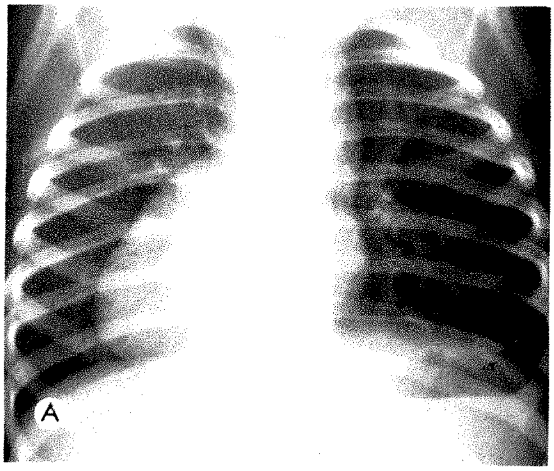


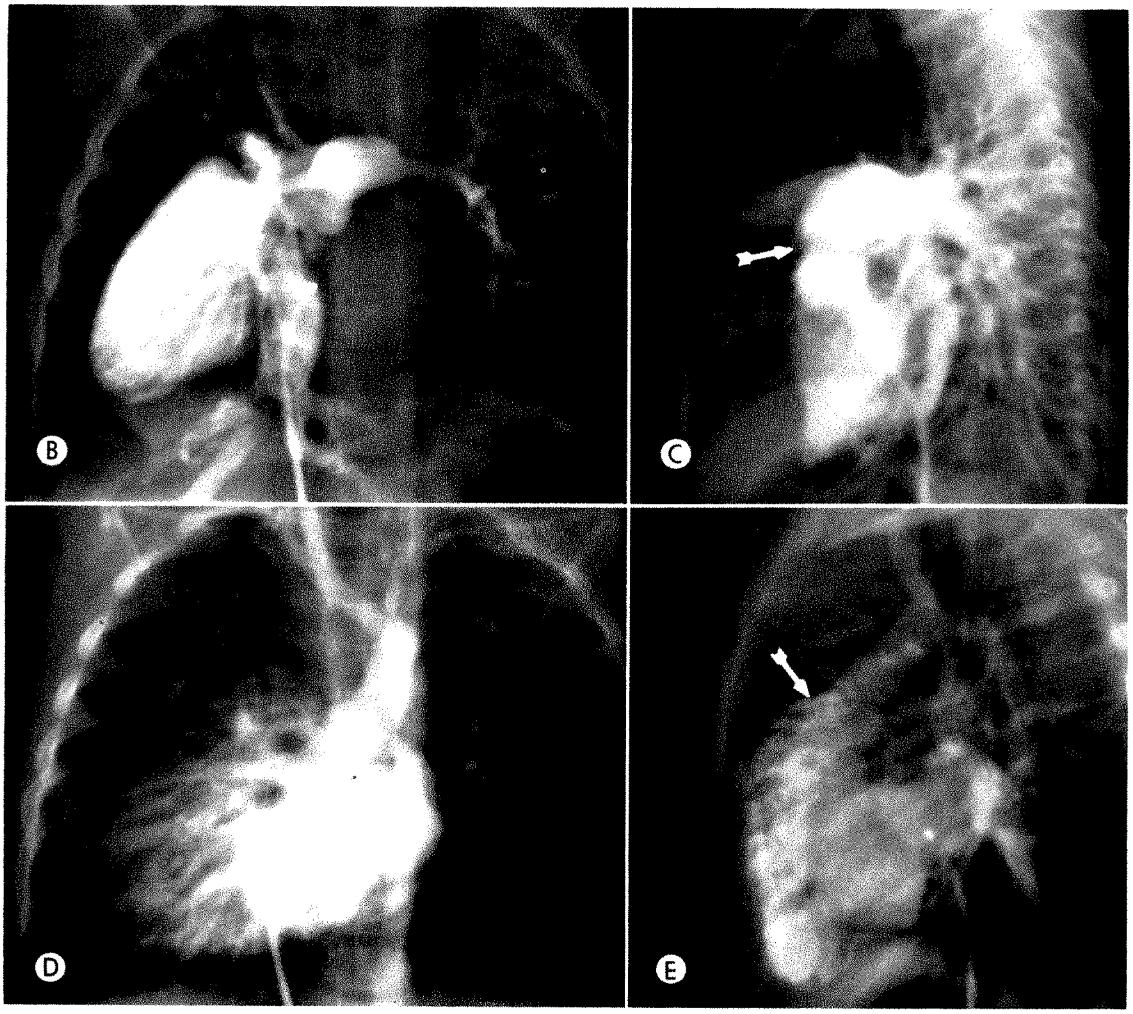
FIG. 6. Type IB. Dextrocardia in situs solitus with discordant L-LOOP. (*A*) Chest roentgenogram showing dextrocardia in situs solitus.

most common type of dextrocardia—comprising 29 per cent of Van Praagh and co-workers'[35,36] series studied at necropsy.

Dextrocardia in situs solitus (Type IA, B or C) has been variously called dextroposition, dextrorotation, dextroversion, isolated dextrocardia, etc.[1,14,19,23,28,35,36,40] The cardiac configuration on the inverted chest roentgenogram seldom resembles the normal, since "mirror-image" dextrocardia in the absence of situs inversus is thought not to exist.[24,35,36]

TYPE IIA DEXTROCARDIA IN SITUS INVERSUS WITH CONCORDANT L-LOOP

Type IIA dextrocardia—"mirror-image" dextrocardia in situs inversus—is the most common type[18,22,29] found in the general



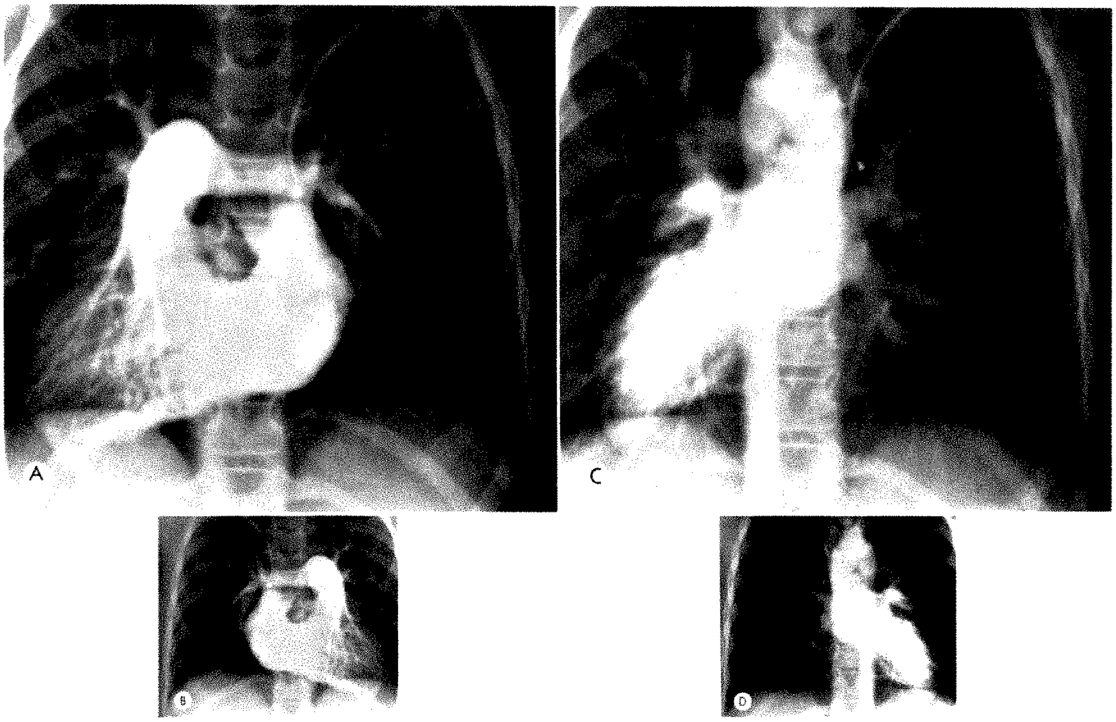


Fig. 7. Type IIA. Dextrocardia in situs inversus with concordant L-LOOP.

(*A*) Frontal angiocardiogram shows opacification of "mirror-image" anatomic right atrium, anatomic right ventricle and pulmonary arteries. The appearance is unfamiliar and thus confusing.

(*B*) When the image is inverted, however, the normal configuration of the anatomic right heart is readily recognized.

(*C*) Frontal angiocardiogram 4 seconds later demonstrates "mirror-image" of anatomic left heart.

(*D*) Again when the image is inverted the normal appearance of an anatomic left atrium and anatomic left ventricle can be appreciated.

adult population (about 1/10,000 to 1/20,000) (Fig. 7, *A–D*). Twelve cases were found in our group of 59 cases with primary dextrocardia. Eight were adults, and only 1 of these has congenital heart disease—pulmonary stenosis and ventricular septal defect. Three of the 4 children had serious congenital heart disease—probably the consequence of selection in our material.

Most individuals with Type IIA dextrocardia have apparently normal longevity. The general incidence of congenital heart

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Fig. 6. (*B* and *C*) Simultaneous frontal and lateral angiocardiograms show the right heart opacified; the right atrium and inferior vena cava are on the right of spine. The opacified right-sided venous ventricle has smooth internal contours, an anterior-superior recess,[3] and a relatively dorsal and caudad origin of the pulmonary valve (arrow)—features indicating an *anatomic left ventricle*. The right atrial appendage protrudes anterior to the pulmonary artery.

(*D* and *E*) Three seconds later when the left heart is opacified, the left atrium is in normal position relative to right atrium. Irregular margins of the coarsely trabeculated, left-sided, ventrally located *anatomic right ventricle* are clearly seen. Ventricular inversion is present. The transposed aorta, characteristic of discordant L-LOOP, arises (arrow) from the infundibulum of the anatomic right ventricle cephalad, ventrad and to the left of the pulmonary artery origin. Ebstein's anomaly deforms left atrioventricular valve.

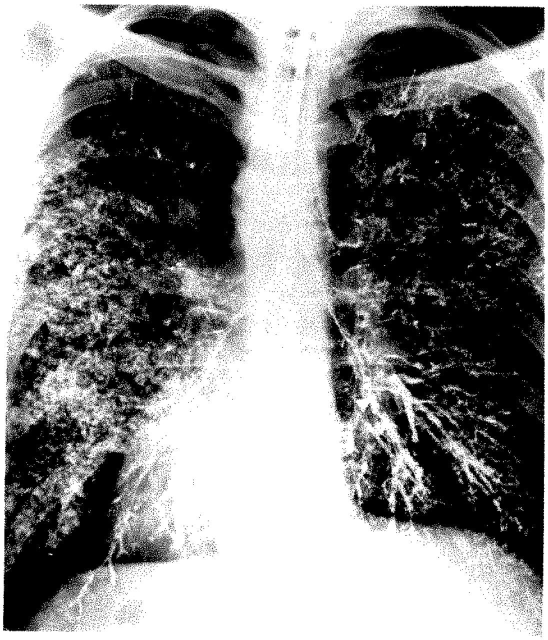

Fig. 8. Type IIA. Dextrocardia in situs inversus with concordant L-LOOP. The bronchogram demonstrates complete situs inversus and bronchiectasis particularly in the retrocardiac region. This 35 year old man had a long history of cough and sputum production. Kartagener in 1933 reported the frequent and unexplained association of situs inversus and "mirror-image" dextrocardia with bronchiectasis and nasal polyps. These patients seldom have congenital cardiac malformations.[20]

disease is low (2–5 per cent). For unknown reasons, 10 to 25 per cent of these people seen in hospitals are found to have bronchiectasis and sinusitis with nasal polyps in addition to situs inversus with dextrocardia.[22] Three of our 12 cases had this syndrome (Kartagener's triad). Necropsies were performed on the 2 who died of complications of the bronchiectasis at ages 8 and 62 years, respectively. The third case is illustrated in Figure 8.

In interpreting the frontal chest roentgenograms of patients with situs inversus, it is useful to invert the roentgenogram, since the resulting image can then be compared with one's general experience dealing with situs solitus individuals. While considering situs inversus as a "mirror-image" of the normal situs solitus is a useful clinical and radiologic concept, it is not literally true since actual right-left reversal or translocation of primordia does not occur. Rather, situs solitus is believed to result from early relative growth dominance of left-sided primordia while situs inversus is considered to result from relative growth dominance of right-sided primordia.[2,5,35]

TYPE IIB DEXTROCARDIA IN SITUS INVERSUS WITH DISCORDANT D-LOOP

We have seen 5 cases of this type, all of which had cyanotic congenital heart disease; 2 of these had double outlet right ventricle. Our oldest patient is a 14 year old boy whose angiocardiogram is shown in Figure 9, *B* and *C*.

This is a rare type of dextrocardia. Not only is situs inversus rare but also discordant loop development is uncommon in situs inversus. Serious intracardiac anomalies are also reported in the few cases recorded in the literature as might be expected in the presence of discordant bulboventricular loop development.

TYPE III DEXTROCARDIA IN INDETERMINATE SITUS (AND ASPLENIA)

Six cases with indeterminate situs were encountered in our series. All had lethal cyanotic congenital heart disease—the oldest living to 2 years. Five had no spleen. Frontal chest roentgenograms showing a symmetric liver, abnormal position of the

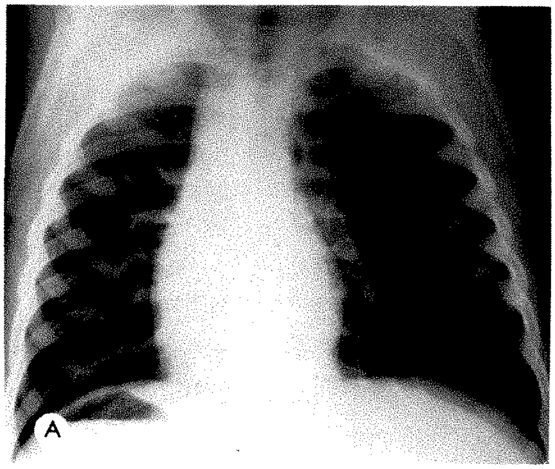

Fig. 9. Type IIB. Dextrocardia in situs inversus with discordant D-LOOP. (*A*) Chest roentgenogram showing dextrocardia in situs inversus.

gastric air bubble and an abnormal cardiac position led to an early consideration of this condition.

Often the spleen is absent when situs is indeterminate. Ivemark,[15] in his definitive study of this asplenia syndrome, found that splenic tissue normally first appears at about 31 days of fetal life, when the atrioventricular canal cushions are in process of fusion and before septation of the bulbotruncal region. The liver is typically symmetric in shape and viscero-atrial situs is indeterminate—there being neither definite situs solitus or situs inversus—a teratologic syndrome of visceral symmetry being present. The commonly associated congenital cardiac defects are:

1. Atrioventricular canal anomalies usually with common atrioventricular valve.
2. Common atrium or atrial isomerism[30]—and a tendency to symmetry of systemic and pulmonary veins.
3. Common ventricle.
4. Anomalous pulmonary venous connection.
5. Transposition of the great vessels.
6. Pulmonary stenosis or atresia.
7. *Dextrocardia.*

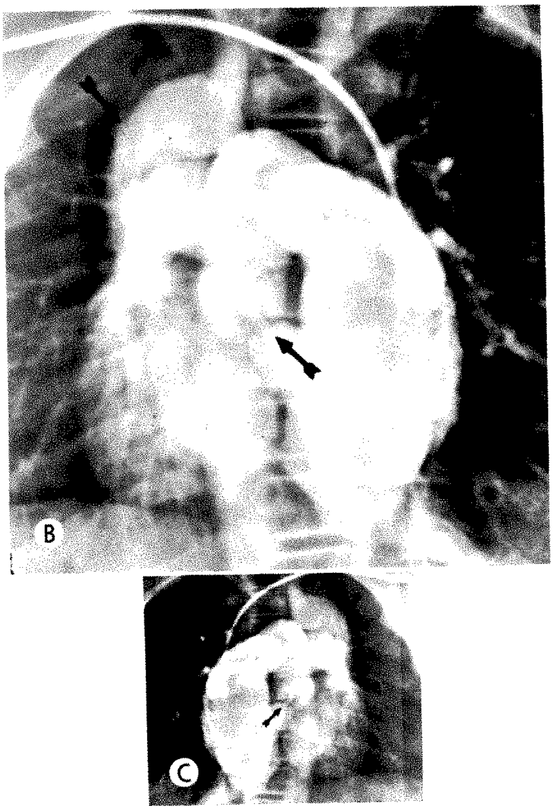

Fig. 9. (*B*) Frontal angiocardiogram of a 14 year old boy shows opacification of anatomic right heart. A jet of contrast material traverses a subvalvular stenosis (lower arrow) at the origin of the pulmonary artery. The ascending aorta (upper arrow) is seen along the right upper heart border, opacified because of a venous to arterial shunt at the level of a ventricular septal defect (discordant D-LOOP). (*C*) Inverted image demonstrating the appearance of the anomaly if situs inversus were not present. Cyanosis and limited exercise tolerance had been present since infancy. A sibling was found at necropsy to have tetralogy of Fallot in situs inversus with "mirror-image" dextrocardia (concordant L-LOOP).

Most examples of the asplenia syndrome have a majority of the above defects, many of which can be shown by angiocardiography. Diagnosis is facilitated by a Wright's stained peripheral blood smear which in the presence of asplenia often reveals red blood cells with retained nuclear fragments called Howell-Jolly bodies. This finding in cyanotic infants with congenital heart disease is considered pathognomonic of asplenia.

Splenic tissue is present in some individuals who have indeterminate viscero-atrial situs and severe congenital cardiac anomalies of the sort found in asplenia.[21] In these cases splenic anomalies and, particularly, multiple spleens are common. On the other hand local abdominal abnormalities in the position of the stomach, spleen, intestines or liver are occasionally found in the presence of a normal heart.[4,20,24]

Rarely, anomalies of cardiac development or position may be encountered which are of more primitive or bizarre nature than those mentioned above (*e.g.*, ectopia cordis, etc.).

COMMENTS CONCERNING TRANSPOSITION OF THE GREAT VESSELS

Transposition of the great vessels can be broadly defined as "any alteration in the

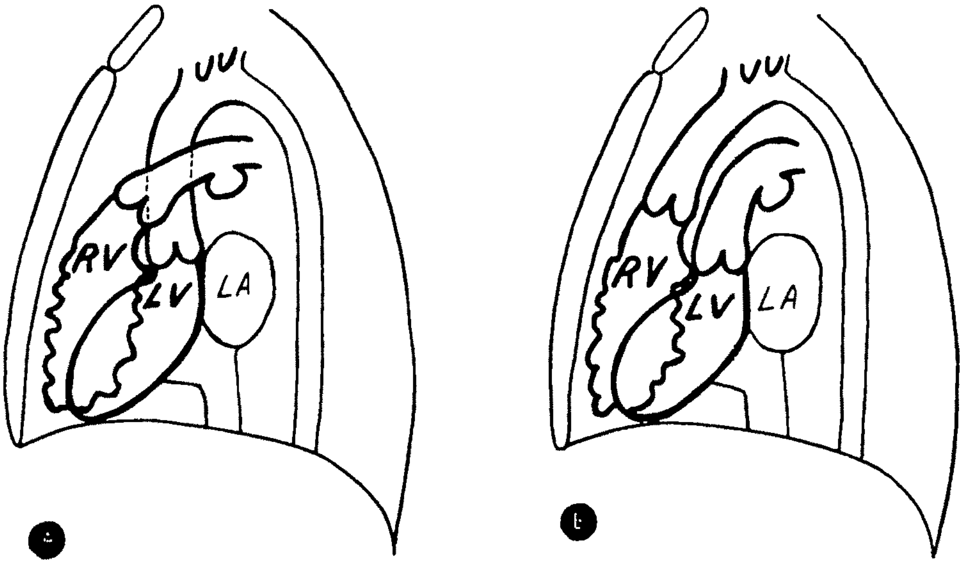

FIG. 10. (*A*) Diagram of normal heart in lateral view. Note the ventral origin of pulmonary artery from the anatomic right ventricle, while the aorta arises more dorsally from the anatomic left ventricle.

(*B*) Diagram of transposition of the great vessels in lateral view. Note that the aorta now arises ventrally from the coarsely trabeculated anatomic right ventricle.

interrelationships of the great arterial trunks and their sites of origin from the heart."[26] *Characteristically in this condition the aorta arises ventrally from the anatomic right ventricle* instead of normally from the anatomic left ventricle (Fig. 10*B*). The pulmonary artery then usually arises more dorsally from the anatomic left ventricle. The ascending great vessels have parallel instead of their normal spiralling courses. The cause is apparently abnormal division of the truncus relative to the ventricles.

In the normal individual (situs solitus with concordant D-LOOP), the pulmonary artery arises from the infundibulum of the right ventricle in a position ventrad, cephalad (and usually to the left) of the site of aortic origin from the left ventricle (Fig. 10*A*). Thus, in frontal projection the aorta normally arises a little to the right of the pulmonary artery.

Transposition of the great vessels only seldom occurs in the presence of the concordant D-LOOP of situs solitus. When this situation does occur, however, the transposed aorta arises from the infundibulum of the right ventricle in a position ventrad, cephalad and also typically slightly to the right of the origin of the transposed pulmonary artery. Moreover, even when a discordant D-LOOP occurs in situs inversus, the origin of the typically transposed aorta is also to the right of the origin of the pulmonary artery.

Thus, the *aortic valve tends to be on the right of the pulmonary valve* whenever D-LOOP type of bulboventricular development is present. Conversely, the *aortic valve tends to be on the left of the pulmonary valve* whenever L-LOOP type of bulboventricular development is present. Despite some exceptions, this relationship is of value in the analysis of frontal angiocardiograms and has been referred to as part of "the loop rule."[34]

Whenever a discordant bulboventricular loop occurs (L-LOOP in situs solitus or D-LOOP in situs inversus) transposition of the great vessels is regularly present (few exceptions are known[17,34]). Several mechanisms have been proposed to account for this transposition which is characteristic of discordant bulboventricular loop development.[8,13,14,27,31,33,39] Anatomic variations in bulbus (or conus) development are numerous and may cause transposition of the great vessels.[38]

Rotation of heart structures secondary to factors such as chamber size, diaphragm position, minor developmental variation, etc., can obviously affect the location of the various cardiac chambers and great vessel origins relative to body surfaces. Such variations explain most inconsistencies in the "loop rule" and those rare cases of transposition of the great vessels (posterior transposed aorta[35]) in which the aortic origin is dorsal to that of the pulmonary artery. These cases can usually be correctly diagnosed by angiocardiography since the relationships of the heart chambers and great vessels to one another are not altered —*e.g.*, the transposed aorta continues to arise from the anatomic right ventricle.

DISCUSSION

Paltauf[23] in 1901 published excellent diagrams of cases representing most types of dextrocardia and recognized that Rokitansky's "corrected" transposition might be present. In 1931 Lichtman[19] thoroughly reviewed the literature on dextrocardia. By 1961 Arcilla and Gasul,[1] in the modern age of pediatric cardiology, were able to present a large personal clinical experience with this entity.

The clear proposals of De la Cruz *et al.*[7,8,9] concerning the embryologic basis for "corrected" transposition of the great vessels and for the potential effects of normal and inverse bulboventricular loop development on congenital dextrocardia made further progress possible. This work aided Espino Vela *et al.*[12] in a splendid study of clinical dextrocardia and helped Rosenbaum[24] in deriving a thoughtful classification of the various spatial alignments of the heart.

The outstanding anatomic and clinical studies of Van Praagh *et al.*[35,36] on a large series of necropsied cases of dextrocardia have provided anatomic evidence of the validity of these previous concepts and have afforded an improved basis for the study of dextrocardia. In this paper, we have drawn heavily upon the work of these earlier workers in providing a classification of dextrocardia. Even at the present time, however, other developmental concepts must also be considered such as: the brilliant phylogenetic theory of Spitzer[27] and Grant's[13] interesting ideas of torsion and cardiac septation. Direct observations on developing human embryos with abnormalities of situs and bulboventricular loop development are very meager. Dekker *et al.*[6] have found a human embryo in which the bulboventricular loop development was inverted (discordant L-LOOP in situs solitus). Shaner has reported similar observations in a pig embryo.[25]

Angiocardiography has afforded an opportunity to study the morphology and function of the various types of dextrocardia. The angiocardiographer has the special advantage in situs inversus of being able to turn over the frontal roentgenograms and immediately view an inverted image of the anomaly. Understanding of the several mechanisms involved in production of dextrocardia is important to intelligent assessment of many cases of congenital heart disease. A further advantage is that the various types of levocardia can be identified as inversions or "mirror-images" of these recognized types of dextrocardia.

Roentgenographic study of primary congenital dextrocardia should include first identification of the viscero-atrial situs (solitus, inversus, or indeterminate), then secondly determination of bulboventricular loop development (D, L or indeterminate). This information permits classification of the dextrocardia. Though the plain chest roentgenogram will indicate the situs development with good reliability, angiocardiography is often necessary for determination of type of bulboventricular loop development. Identification of the anatomic right and left ventricles in relation to the anatomic right and left atria will indicate whether bulboventricular loop development is concordant or discordant. If the aorta arises on the right of the pulmonary artery, a D-LOOP is probably present; if on the left, an L-LOOP.[35] If transposition of the great vessels is not present, discordant bulboventricular loop development is virtually excluded. Once the type of dextrocardia is appreciated, other congenital cardiac anomalies are more readily identified.

Of course, a clinically normal heart in situs inversus with normal configuration on the inverted chest roentgenograms can be classified IIA without further study. Likewise, clinically normal cardiac function in dextrocardia in situs solitus, with normal heart size and lung fields on the chest roentgenogram, is very likely Type IA dextrocardia, *i.e.*, normal situs and loop development with failure of leftward shift of the ventricles, since Types IB and IC nearly always have severe associated cardiac anomalies. Occasionally, precise diagnostic classification may remain uncertain despite angiocardiography in dextrocardia with complex associated anomalies. Knowledge concerning the expected consequences of aberrations in cardiac development, however, increases the possibilities for accurate diagnosis.

SUMMARY

A comprehensive and useful classification of dextrocardia is presented. It is based on the viscero-atrial situs (solitus, inversus, or indeterminate) and on the direction of bulboventricular loop bending during development (D or L-LOOP).

Roentgenographic examination and, particularly, angiocardiography are essential to the recognition of the various types of dextrocardia. Precise anatomic diagnosis is indicated for the frequently associated congenital cardiac malformations, many of which are now amenable to surgical correction.

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OBLIQUE LEFT VENTRICULOGRAPHY IN ENDOCARDIAL CUSHION DEFECT, GRADE III*

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RAPID advances have been made in the operative treatment of cardiac disease. Inevitably, attention is now being directed to correction of the more complex cardiac malformations. These and future surgical advances are dependent on the increasing sophistication of preoperative diagnosis of cardiac disease. Cardioangiography is the forerunner in this field.

In several recent publications,[2,4,7] especially those of Baron and his colleagues, the cardioangiographic features of endocardial cushion defects, including the ostium primum type of atrial septal defect, are described. The endocardial cushion defects represent a spectrum of related malformations of atrial and ventricular septation and atrioventricular valve leaflet development.[12] The purpose of the authors is to demonstrate the cardioangiographic characteristics of the most severe type of this related group of malformations, endocardial cushion defect, Grade III (complete form of persistent common atrioventicular canal).

EMBRYOLOGY

In the primitive heart, there is free communication between the undivided atrial and ventricular cavities by means of the atrioventricular canal (Fig. 1*A*).[2,11] During the second month of development, the septum primum arises from the posterosuperior wall of the common atrial chamber and grows in the direction of the atrioventricular canal, providing a partition between the atria (Fig. 1*B*). The division is incomplete in that a residual communication, the ostium primum, persists at the anteroinferior border of this septum. At about the same time that the atrium is being divided into right and left chambers, two thickenings appear which project from the superior and inferior walls of the atrioventricular canal. These opposing masses, the endocardial cushions, fuse with each other to divide the atrioventricular canal into a right and left orifice (Fig. 1*C*).[2] The septum primum then joins with the endocardial cushions to obliterate the ostium primum. As this is occurring, a new opening appears more cephalad in the septum primum, the ostium secundum. There is thus assured a direct pathway for blood from the right atrium to the left atrium, an essential feature of the fetal circulation. This is in contrast to the complete partition of the ventricles which occurs early in embryonic development. A second septum, the septum secundum, develops just to the right of the septum primum (Fig. 1*D*).[11] The septum secundum does not develop into a complete septum, but rather has an opening, the foramen ovale, which, with the septum primum, forms a one-way valve, permitting blood to go from the right to the left atrium but not in reverse.

At about the same time that the septum primum appears another septum, the interventricular septum, develops from the apex of the ventricle and grows toward the endocardial cushions. This results in nearly complete division of the ventricles. Final contributions to closure of the remaining interventricular foramen come from the interventricular septum itself, the endocardial cushions, and the conus ridges. These three structures form the membranous interventricular septum.[15]

The posterior mitral leaflet, and the

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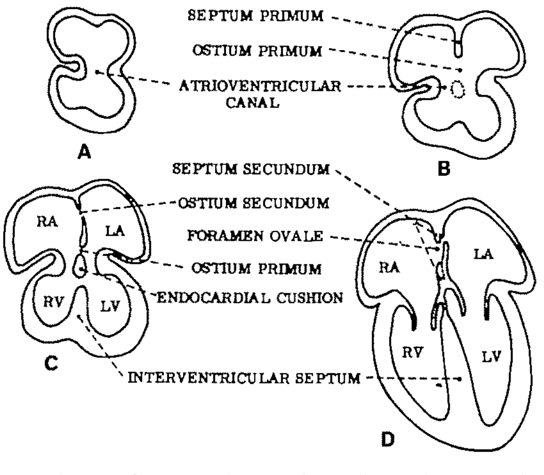

FIG. 1. Diagrammatic representation of the septation of the embryonic heart. (*A*) 4–5 mm. stage. Free communication between the undivided atrial and ventricular cavities by means of the atrioventricular canal. (*B*) 6–7 mm. stage. The septum primum is seen growing in the direction of the common atrioventricular canal. The ostium primum persists at the anteroinferior border of this septum. (*C*) 8–9 mm. stage. The endocardial cushions are seen dividing the atrioventricular canal into a right and a left orifice. The septum primum is joining with the endocardial cushions to obliterate the ostium primum. A new opening has appeared in the septum primum, the ostium secundum. (*D*) 100 mm. to birth. The septum secundum has developed just to the right of the septum primum. An opening in this septum, the foramen ovale, forms a one-way valve with the septum primum to permit blood to enter the left atrium from the right atrium but not in reverse. Complete development and closure of the interventricular septum have occurred. RA = right atrium; RV = right ventricle; LA = left atrium; LV = left ventricle.

anterior and posterior leaflets of the tricuspid valve are formed from the endocardial tissue at the margins of the atrioventricular canal. The anterior leaflet of the mitral valve and the septal leaflet of the tricuspid valve originate from the superior and inferior endocardial cushions. Since these cushions play a major role in atrial and ventricular septation as well as in development of the anterior mitral and septal tricuspid leaflets, maldevelopment can result in a wide range of related congenital cardiac anomalies.

CLASSIFICATION

This spectrum of malformations can be divided into 6 groups:

(1) Endocardial cushion defect, Grade I[3] (persistent common atrioventricular canal, partial form,[15] persistent ostium primum with cleft mitral valve[12]). Here, there is a defect in the lower portion of the atrial septum and upper portion of the ventricular septum. The attachment of the anterior mitral leaflet to the crest of the remaining ventricular septum prevents interventricular communication. The upper margin of the defect is crescentic. In a normal heart the line of attachment of the anterior mitral leaflet is in the region of the deficient septum. In the presence of this anomaly, however, the cleft anterior mitral leaflet is attached to the ventricular septum more toward the cardiac apex. Failure of fusion of the superior and inferior endocardial cushions on the left side results in the cleft anterior mitral leaflet. The tricuspid valve is not cleft and rarely the anterior mitral leaflet is not cleft.

(2) Endocardial cushion defect, Grade II[3] (persistent common atrioventricular canal, transitional type,[16] persistent ostium primum with cleft mitral and tricuspid valves[12]). Clefts are present in both the anterior mitral and the septal tricuspid valve leaflets but a "narrow bridge" of valve tissue joins the superior and inferior halves of each cleft just above the ventricular septum. The interatrial communication is similar to that seen in the Grade I defect. The membranous interventricular septum may be deficient.

(3) Endocardial cushion defect, Grade III[3] (persistent common atrioventricular canal, complete form[15]). There is complete lack of fusion of the endocardial cushions. The defect in the atrial septum resembles the Grade I defect. A common cleft divides both the anterior mitral and septal tricuspid leaflets. There is an interventricular communication between the common atrioventricular valve and the incomplete ventricular septum. The size and appearance of this defect in the ventricular septum are variable but usually there is a sizable deficiency.

(4) Endocardial cushion defect without interatrial communication and with interventricular septal defect[14] (persistent common atrioventricular canal type of ventricular septal defect[10]). Most cases have clefts or deformities of one or both of the atrioventricular valve leaflets derived from the endocardial cushions. There is failure of closure of the interventricular foramen. This results from lack of fusion of the elements that complete development of the interventricular septum—the conus ridges, endocardial cushions, and muscular ventricular septum.

(5) Endocardial cushion defect with left ventricle-right atrial communication and cleft septal leaflet of the tricuspid valve.[6]

(6) Endocardial cushion defect with cleft anterior mitral leaflet but with intact atrial and ventricular septa.[13]

CARDIOANGIOGRAPHY

Cinecardioangiograms of 17 cases of endocardial cushion defect, Grade III, were reviewed as a basis for this study. Left ventricular injections were performed in 9 of these cases. All 9 demonstrated deformity of the outflow portion of the left ven-

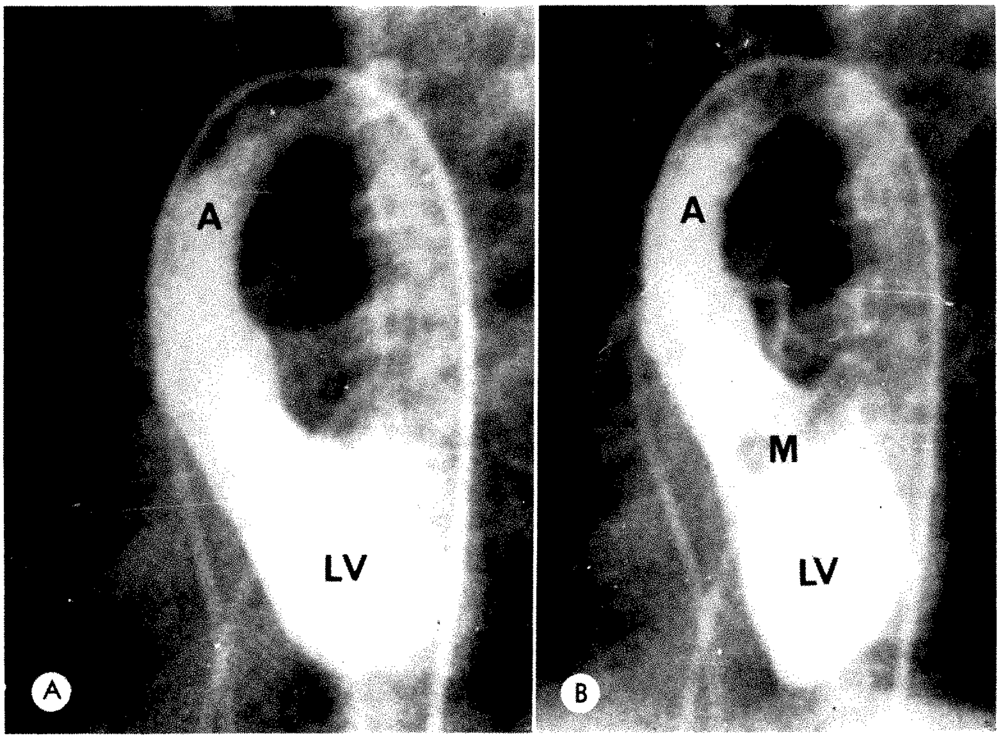

Fig. 2. Nine year old female with secundum atrial septal defect and normal mitral valve and left ventricle. Left anterior oblique cineroentgenograms with injection of contrast medium into the left ventricle. (*A*) Systolic phase. Smooth medial wall of the left ventricle (LV). No distortion of left ventricular outflow. (*B*) Diastolic phase. The normal mitral valve (M) is seen opening into the left ventricle (LV) lateral to the ventricular septum. There is preservation of continuity of the medial wall of the ventricle. Catheters are in the left ventricle, left atrium, and pulmonary artery. A = aorta

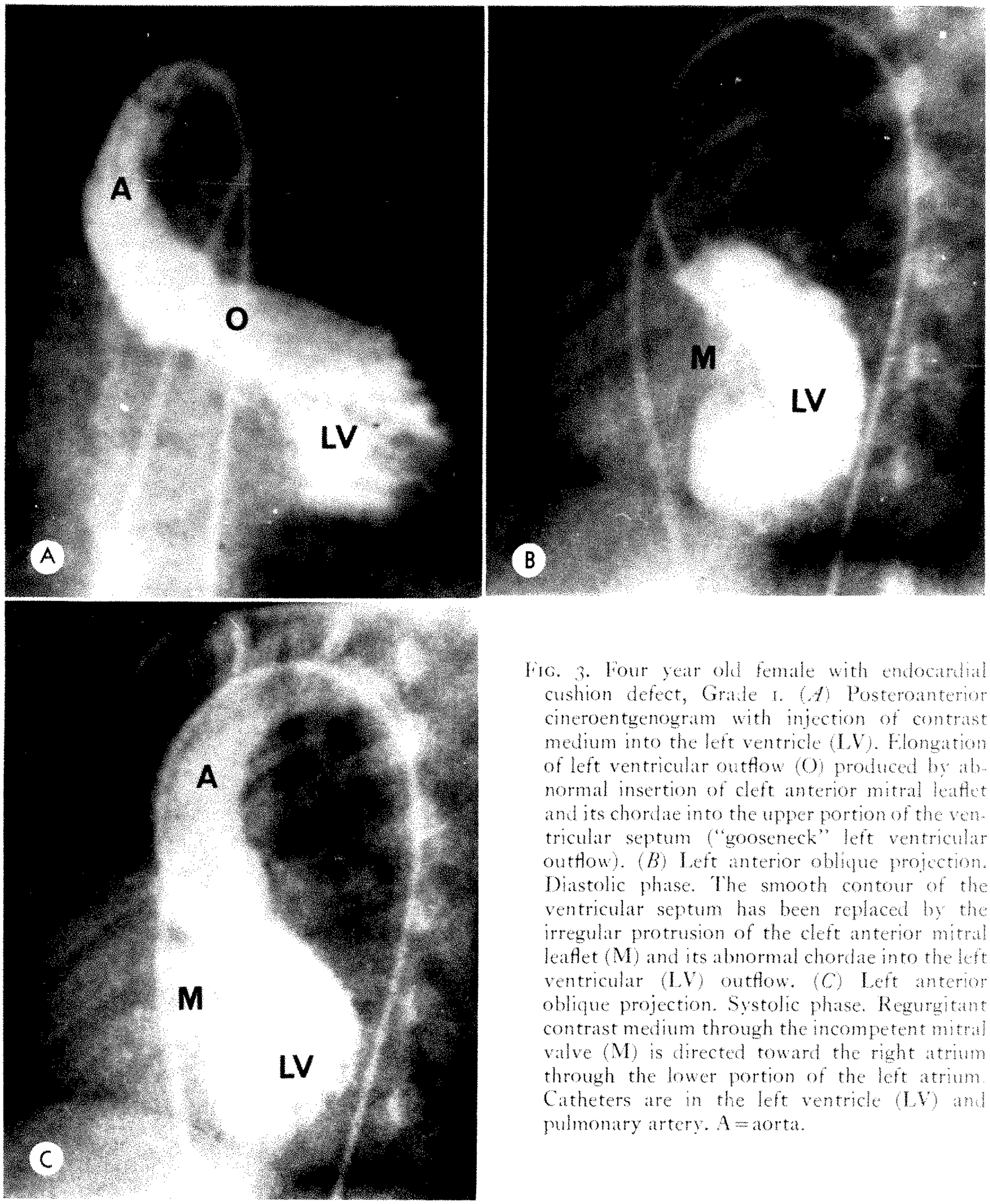

Fig. 3. Four year old female with endocardial cushion defect, Grade I. (*A*) Posteroanterior cineroentgenogram with injection of contrast medium into the left ventricle (LV). Elongation of left ventricular outflow (O) produced by abnormal insertion of cleft anterior mitral leaflet and its chordae into the upper portion of the ventricular septum ("gooseneck" left ventricular outflow). (*B*) Left anterior oblique projection. Diastolic phase. The smooth contour of the ventricular septum has been replaced by the irregular protrusion of the cleft anterior mitral leaflet (M) and its abnormal chordae into the left ventricular (LV) outflow. (*C*) Left anterior oblique projection. Systolic phase. Regurgitant contrast medium through the incompetent mitral valve (M) is directed toward the right atrium through the lower portion of the left atrium. Catheters are in the left ventricle (LV) and pulmonary artery. A = aorta.

tricle as described by Baron *et al.*,[2] but, in addition, 5 demonstrated findings permitting diagnosis of endocardial cushion defect, Grade III. Four of these 5 cases were proved by postmortem examination.

For almost a decade, single plane selective cinecardioangiography has been performed at this institution in almost every case undergoing cardiac catheterization. The position of choice for demonstration of defects in the ventricular septum has been the 60° left anterior oblique position. Although previous authors have emphasized left ventricular cardioangiography in frontal and lateral projections, we have found the left anterior oblique projection to be almost as good in demonstrating the deformity associated with endocardial cushion defects. This deformity results from attachment of the cleft, deformed, anterior mitral leaflet to the left ventricular aspect of the ventricular septum. In addition, since a major portion of the ventricular septum is seen in profile in this projection, we have been able to differentiate between endocardial cushion defects, Grades I and III, by demonstration of the interventricular communication and by observing the large common atrioventricular valve opening over the deficient ventricular septum.

In the normal heart in the left anterior oblique position, the medial aspect of the left ventricle is formed by the muscular septum below and membranous septum above. This margin of the left ventricle is identifiable in systole and diastole as the uninterrupted medial wall which is smooth in its upper portion and slightly irregular below (Fig. 2, *A* and *B*). During the diastolic phase of the left ventriculogram, the mitral valve can be identified by the dilution effect of unopacified left atrial blood entering the opacified left ventricle. The integrity of the left ventricular aspect of the ventricular septum persists. In the presence of endocardial cushion defects, Grades I, II, and III, there is a consistent deviation from the normal configuration of this aspect of the ventricular septum (Fig. 3, *A*, *B* and *C*). This results from the fact that the abnormal cleft anterior mitral leaflet is immediately adjacent to the superoposterior aspect of the ventricular septum. The sharp, well defined left ventricular aspect of the ventricular septum is replaced by an irregular scalloped appearance. In addition, during ventricular diastole, the cineroentgenographic sequence

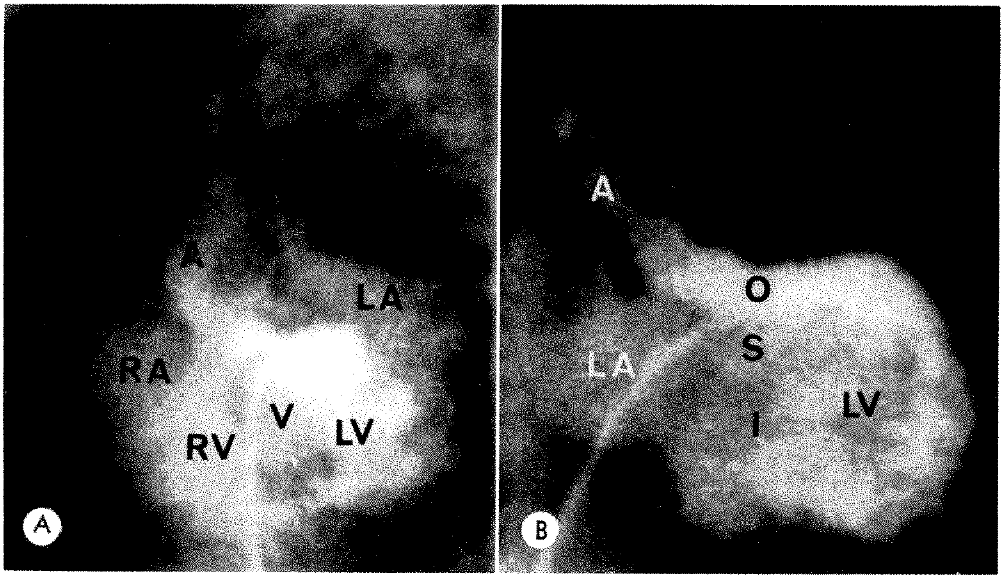

Fig. 4. Six week old female with endocardial cushion defect, Grade III (postmortem proof). (*A*) Left anterior oblique cineroentgenogram with injection of contrast medium into the left ventricle. Diastolic phase. The large common atrioventricular valve (V) is seen opening over the region of the interventricular septum. The inflow of unopacified atrial blood produces this filling defect, which is probably larger than the true atrioventricular valve orifice. RA = right atrium; RV = right ventricle; LA = left atrium; LV = left ventricle; A = aorta. (*B*) Right anterior oblique cineroentgenogram with injection of contrast medium into left ventricle (LV). Diastolic phase. The superior portion of the cleft common atrioventricular valve (S) has moved upward to produce elongation of the left ventricular outflow (O). The inferior portion of the cleft valve (I) is also seen. The left ventricular injections were made by a catheter which traversed the inferior vena cava, right atrium and left atrium (LA) to reach the left ventricle. A = aorta.

shows the opening mitral valve merging with the irregular margin of the interventricular septum. When mitral regurgitation is present, which is frequently the case, contrast medium may opacify the left atrium. More often, however, the opaque material seems to be directed preferentially toward the right atrium, skirting through the lower portion of the atrial septum and only slightly opacifying the left atrium.

By left ventricular cinecardioangiography in the left anterior oblique projection, the deformity of the left ventricular aspect of the ventricular septum in endocardial cushion defects, Grades I, II, and III, is indistinguishable. Frequently in all 3 there is mitral regurgitation with opacification of one or both atria. In the presence of a sizable interventricular communication (Grade III), there is early opacification of the right ventricle or, if mitral regurgitation is also present, opacification of the right ventricle and one or both atria. The ventricular septum is then seen in profile. When the next ventricular diastole occurs, the unopacified or slightly opacified atrial blood can be seen entering the ventricles by the large common atrioventricular valve which opens over the ventricular septum (Fig. 4, *A* and *B;* and 5). If the ventricular septal defect is small or guarded by the cleft abnormal leaflets, this finding will be less obvious or absent. Since many cases of endocardial cushion defect, Grade III, have large interventricular communications, it should be possible to frequently identify the common atrioventricular valve by left ventricular cinecardioangiography in the left anterior oblique projection.

The characteristic deformity of the outflow portion of the left ventricle, as seen in the 3 major types of endocardial cushion defects (Grades I, II, and III), has not been established in endocardial cushion defect without interatrial communication and with ventricular septal defect, endocardial cushion defect with left ventricle-right atrial communication and endocardial cushion defect with cleft anterior mitral leaflet but with intact atrial and ventricular septa.

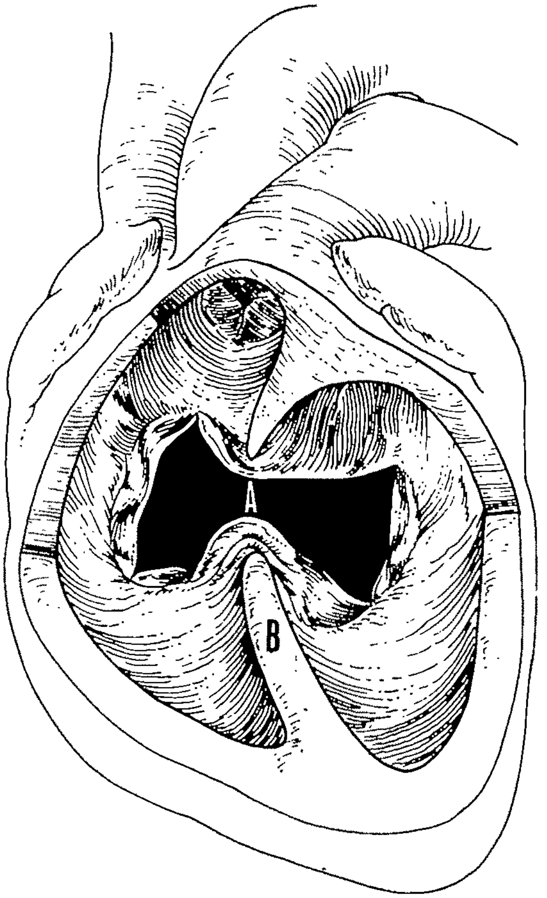

Fig. 5. Diagrammatic representation of endocardial cushion defect, Grade III, in the left anterior oblique projection. The common atrioventricular valve (A) is seen opening over the deficient interventricular septum (B).

DISCUSSION

The differentiation of endocardial cushion defects, Grades I and III, is of great importance. Mortality for repair of the Grade I defect is approximately 16 per cent as opposed to 73 per cent or greater mortality for the Grade III variety.[1,9] It has been recommended that total correction not be performed in infancy in instances of the Grade III defect, but instead that a pulmonary artery banding be performed. In addition complete repair of this malformation may require atrioventricular valve replacement.

Many patients with endocardial cushion defect, Grade III, have in addition defects in the region of the fossa ovalis. Associated anomalies among others are patent ductus arteriosus, double mitral valve, and subaortic stenosis. The latter may result from adherence of the anterior mitral leaflet to the edge of the deficient ventricular septum during ventricular systole.[13]

Thirty to 50 per cent of cases with the Grade III malformation are found in mongols.[5,8]

SUMMARY

The embryology and anatomy of the group of cardiac malformations referred to as endocardial cushion defects have been reviewed.

Specific cardioangiographic features in frontal and lateral projections, described previously, permit recognition of the vast majority of cases that fall within this group. The cinecardioangiographic characteristics of oblique left ventriculography in differentiation of the 2 most common forms of endocardial cushion defects have been demonstrated. Brief reference has been made to the importance of this differentiation.

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ANGIOCARDIOGRAPHIC FINDINGS IN VENTRICULAR ANEURYSM DUE TO ARTERIOSCLEROTIC MYOCARDIAL INFARCTION*

REPORT OF 11 CASES

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ANGIOCARDIOGRAPHY for visualization of ventricular aneurysms due to myocardial infarction was first used in 1939 (Fig. 1). Even though clinical diagnosis of ventricular aneurysm lags well behind the pathologic[1,8,9,12,15,20,22,28] and angiocardiography, especially cineangiocardiography, has been generally accepted as the definitive way to establish the diagnosis, only a few papers have appeared on this subject.[2,3,6,7,9,12,13,17,18,23] This is probably because treatment of postinfarctional aneurysms with open-heart techniques has only recently come into use.[3–5,10,12] Furthermore, even though the diagnosis of ventricular aneurysm is often missed, once suspected, conventional roentgenoscopy and roentgenography are usually sufficient for diagnosis.[15,22] In addition, there is understandable reluctance to catheterize and inject contrast material into a cardiac chamber suspected of containing mural thrombi of a seriously ill patient. For this reason, a series of 11 cases of left ventricular aneurysm following myocardial infarction visualized via the intravenous route is presented. No ill effects, however, have been reported following selective ventriculography via direct ventricular[2,17,23] or transatrial puncture[11,12] and retrograde aortography.[10,12,18]

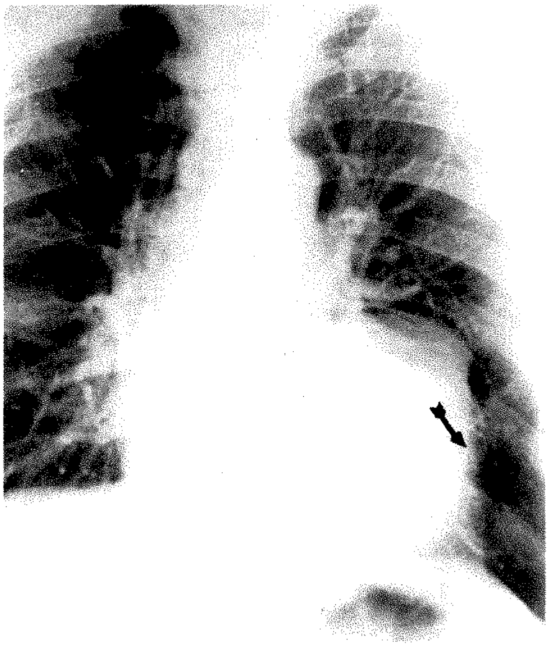

FIG. 1. Case 1. Frontal angiocardiogram, made in 1939, showing the thinned lateral wall of a left ventricular aneurysm (arrow) with a huge thrombus above it.

METHODS AND MATERIALS

All patients had complete clinical and laboratory evaluation which included electrocardiography, conventional roentgenography in all positions, and roentgenoscopy. Intravenous angiocardiography was at first performed manually[19] and later by pressure injection.[27] Two 14 by 17 inch films per injection using the stereocassette apparatus[7,19] were exposed in the first 3 patients reported in this series. Later, beginning in 1953, when the Fairchild multiple film 12 by 12 inch magazine became available,[25] serial angiocardiograms of the cardiovascular system were obtained. The last 5 patients had simultaneous, biplane, frontal and

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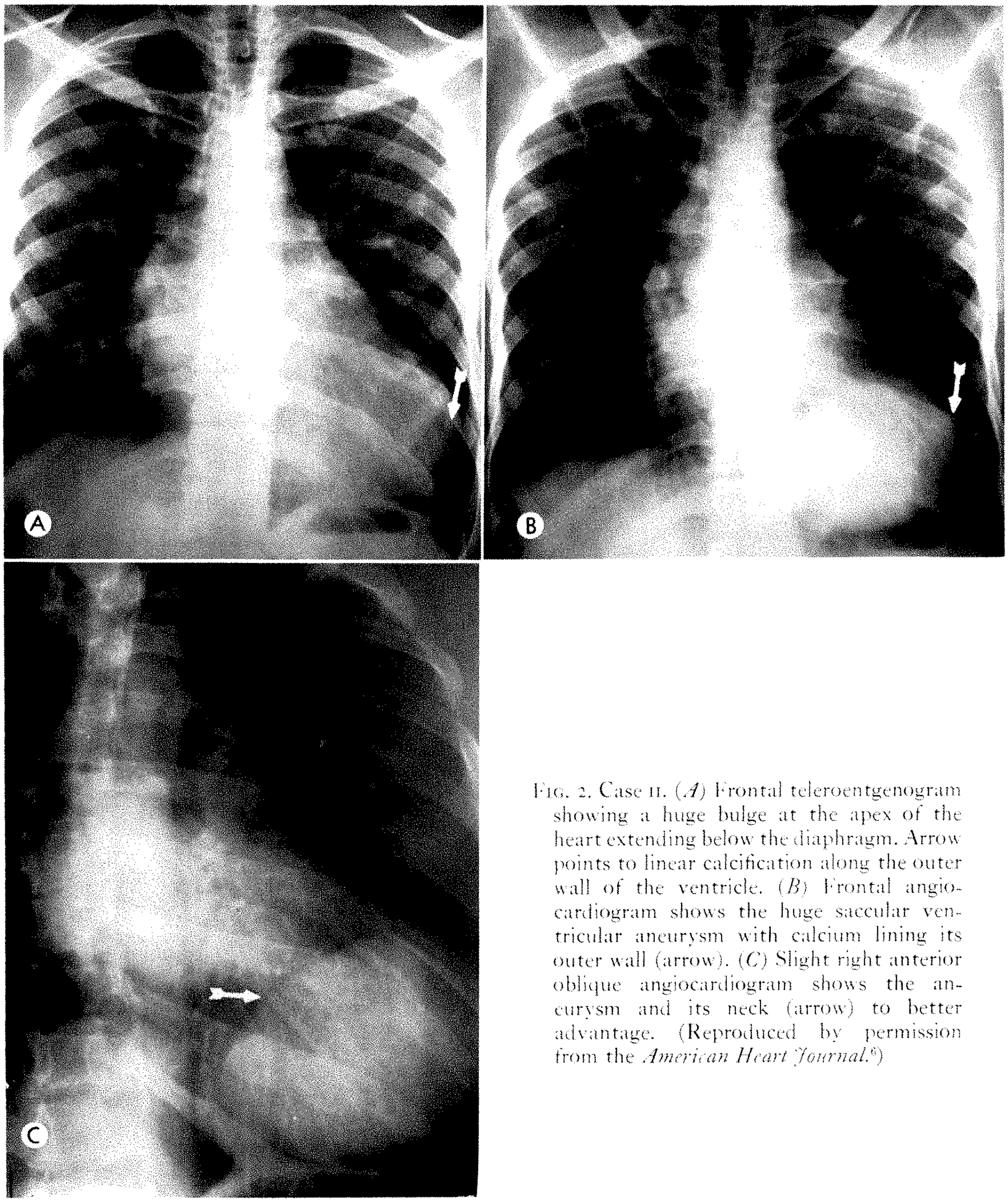

Fig. 2. Case II. (*A*) Frontal teleroentgenogram showing a huge bulge at the apex of the heart extending below the diaphragm. Arrow points to linear calcification along the outer wall of the ventricle. (*B*) Frontal angiocardiogram shows the huge saccular ventricular aneurysm with calcium lining its outer wall (arrow). (*C*) Slight right anterior oblique angiocardiogram shows the aneurysm and its neck (arrow) to better advantage. (Reproduced by permission from the *American Heart Journal*.[6])

lateral, 14 by 14 inch film studies (Schönander apparatus). Cineangiocardiographic intravenous studies were also made of 3 patients. All except 1 of the patients (Case II) were Caucasian. Only 1 patient was a woman (Case XI). The ages varied from 29 years (Case II) to 64 years; the average was 54 years.

REPORT OF CASES

Case I. *Anterolateral myocardial aneurysm.* A 56 year old Caucasian man was admitted on July 20, 1939, to Lenox Hill Hospital with complaints of recurrent attacks of severe oppressive precordial pain and dyspnea. These followed myocardial infarction sustained 4 years earlier. Roentgenograms of the chest revealed a ventricular aneurysm. On physical examination,

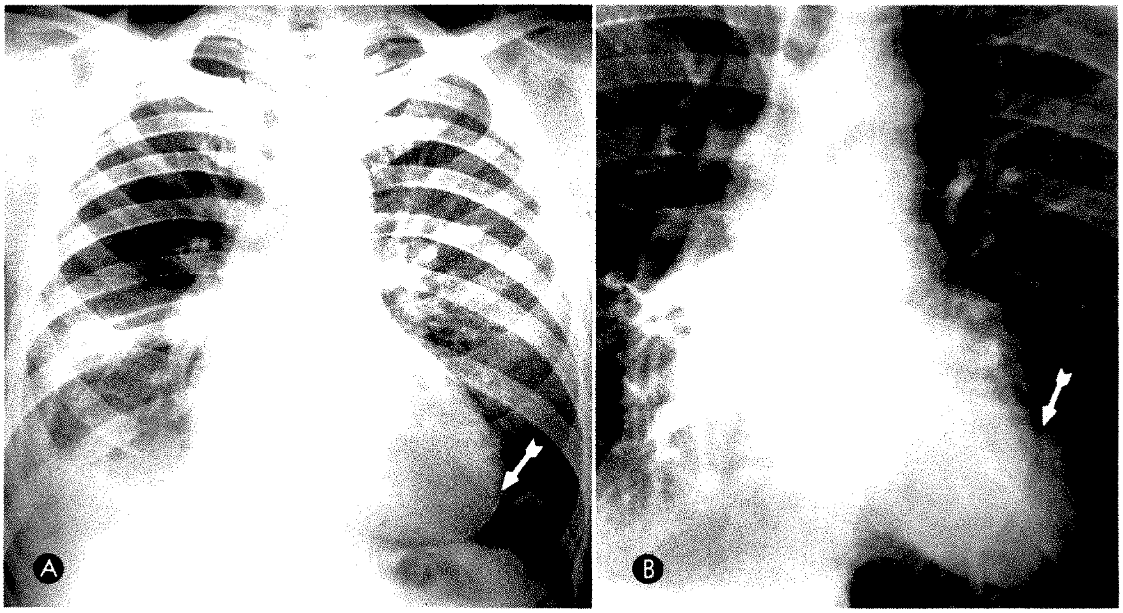

FIG. 3. Case III. (*A*) Frontal teleroentgenogram showing enlargement of the heart and linear calcification along the outer wall of the left ventricle (arrow). (*B*) Frontal angiocardiogram shows the neck of the apical aneurysm (arrow).

moist rales were heard at the left base. The heart was enlarged, the rate was regular, and the blood pressure was 145/110 mm. Hg. A blowing systolic murmur was heard at the apex. The liver was tender and enlarged and was felt 4 cm. below the right costal margin; there was no edema of the legs. The electrocardiogram showed left axis deviation and anterolateral myocardial infarction. Angiocardiography, performed on July 22, 1939, disclosed a huge left ventricular aneurysm lined by thrombus in the superior aspect of the left ventricle, but with marked thinning and calcification of the wall along the lower left outer border of the heart wall (Fig. 1).

CASE II. *Huge apical saccular ventricular aneurysm.* A 29 year old Negro man (N.Y.H. No. 572 295, previously published[6]) was referred for angiocardiography on June 20, 1950. He had had exertional dyspnea and precordial pain for 3 years following severe chest pain which had been erroneously diagnosed as pneumonia. The heart was enlarged, a rough systolic murmur was heard over the pulmonic area and softer systolic and diastolic murmurs were heard at the apex. There was a heaving apical impulse and the blood pressure was 110/50 mm. Hg. Electrocardiograms showed left axis deviation and old myocardial infarction of the anterolateral and posterior portions of the left ventricle. Roentgenograms of the chest revealed marked left ventricular bulging almost reaching the left thoracic wall and extending below the left diaphragm (Fig. 2*A*). Roentgenoscopy revealed paradoxical pulsations of the aneurysm. Angiocardiograms in frontal and right anterior oblique views showed a rounded 7 cm. in diameter saccular aneurysm arising from the anterior wall of the left ventricle (Fig. 2, *B* and *C*).

CASE III. *Anterolateral myocardial aneurysm.* A 61 year old diabetic Caucasian man (N.Y.H. No. 278 898) had a history of hypertension and myocardial infarction 10 years prior to admission on May 1, 1951. Despite treatment with digitalis and diuretics, dyspnea on exertion developed. On examination, the heart was found to be enlarged and a systolic (Grade 3) murmur transmitted to the left axilla was heard at the apex. The liver was enlarged 5 cm. below the right costal margin; there was no edema of the legs. The modified circulation time was prolonged, 19 seconds (the average normal circulation time is 8 to 10 seconds). The blood pressure was 140/75 mm. Hg. The electrocardiogram showed left axis deviation, left bundle branch block (discordant type), old antero-apical myocardial infarction, and regular sinus rhythm at a rate of 88. The fasting blood

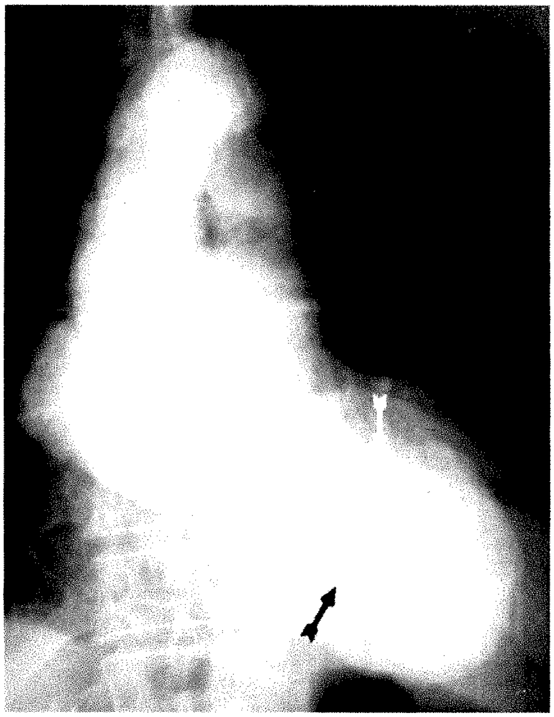

Fig. 4. Case iv. Frontal angiocardiogram showing the huge saccular outpouching of an apical cardiac aneurysm. Arrows point to the neck of the aneurysm.

sugar was elevated. A roentgenogram of the chest showed enlargement of the heart, especially the left ventricle which contained linear calcifications (Fig. 3*A*) and pulsated paradoxically. The right hemithorax also contained a pleural effusion. Angiocardiograms, on May 15, 1951, showed a left ventricular aneurysm along the anterolateral wall of the left ventricle (Fig. 3*B*). The patient died at home of congestive heart failure on May 6, 1952. Autopsy was not obtained.

Case iv. *Huge antero-apical ventricular aneurysm; explored and found inoperable.* A 59 year old Caucasian man (N.Y.H. No. 772 028) was admitted on July 10, 1957, for treatment of ventricular aneurysm. He had experienced sudden onset of epigastric heaviness on February 2, 1957. An electrocardiogram was made and an anterolateral wall myocardial infarction was diagnosed. He was then admitted to a hospital where he was kept in bed and given anticoagulants. Previous to hospitalization, he began losing weight, became more dyspneic, and a month before admission blood-streaked sputum was observed. Roentgenoscopy and roentgenography demonstrated a huge ventricular aneurysm.

Examination revealed mild dyspnea and fine rales were heard at both bases of the lungs. The heart was enlarged and a systolic (Grade 2) murmur was heard at the base of the heart. The liver was enlarged 2 cm. below the right costal margin; there was no edema of the legs. The blood pressure was 115/82 mm. Hg. An electrocardiogram showed regular sinus rhythm, rate of 94, left axis deviation, and antero-apical myocardial infarction. The chest roentgenogram showed marked enlargement of the heart with bulging of the left ventricle. Roentgenoscopy revealed absence of pulsations along the left ventricular bulging area.

Angiocardiograms, on July 11, 1957, showed a huge left ventricular aneurysm surrounded by thrombus (Fig. 4). During a 10 second interval hardly any contractions of the left ventricular aneurysm were present. At exploratory thoracotomy and pericardiotomy on July 17, 1957, almost the entire left heart consisted of the ventricular aneurysm with only a small normal functioning portion. Excision of the aneurysm appeared inadvisable. He did well following operation and was discharged on August 2, 1957. His record contains a note that he was alive on December 12, 1962, and had been admitted to another hospital.

Case v. *Huge anterolateral ventricular aneurysm; good cardiac hemodynamics precluded surgery.* A 39 year old Caucasian man (N.Y.H. No. 811 770) was admitted for angiocardiography and cardiac catheterization on December 8, 1958, in order to determine whether surgery should be performed for a ventricular aneurysm. He experienced episodes of palpitation for 5 years, averaging 2 attacks per year. Three months before admission he was admitted to another hospital because of this and, following serial electrocardiograms, a diagnosis of myocardial infarction was made. After a 6 weeks' period of bed rest, a large left ventricular aneurysm was seen in the roentgenogram of the chest and transfer to this hospital was arranged. On examination, the patient was found to be well developed, well nourished and in no distress. The heart was enlarged to the left axillary line. There were no murmurs. The blood pressure was 135/85 mm. Hg. The electrocardiogram showed anterior apical myocardial infarction and regular sinus rhythm with a rate of 81.

Angiocardiograms on December 9, 1958, showed a huge left ventricular aneurysm (Fig. 5). No movement of the aneurysm was seen during roentgenoscopy. Cardiac catheterization on December 11, 1958, showed slightly decreased cardiac output during rest and exercise. The pulmonary wedge and right ventricular pressures were normal at rest, becoming only slightly increased after exercise. Although moderate impairment of left ventricular filling was found, the striking preservation of cardiac function, despite the myocardial infarction and aneurysm, precluded surgery and the patient was discharged.

Case vi. *Posterobasilar myocardial aneurysm surrounded by multiple calcifications.* A 62 year-old Caucasian man (N.Y.H. No. 370 600) had a history of an acute posterior basal myocardial infarction in 1940, which was followed by angina and dyspnea on exertion. Four years later, in 1944, a left ventricular aneurysm with paradoxical pulsations was noted. In 1958, intravenous angiocardiograms showed a large left ventricular aneurysm with thrombus and calcification of the wall. Cardiac catheterization, also in 1958, showed normal pulmonary capillary pressure at rest with moderate elevation after exercise. The pulmonary pressure was also elevated at rest and increased after exercise. Cardiac surgery was not advised, since repair of the aneurysm was not expected to cause a significant increase in the cardiac output.

The patient was readmitted on March 9, 1965, 7 years later. At this time he complained of dyspnea on walking up 1 flight of stairs as well as nocturnal paroxysmal dyspnea. Chest pain had subsided and there was no edema of the legs. Physical examination revealed obesity and enlargement of the heart. An apical systolic murmur (Grade 4–6) was heard; the blood pressure was 152/80 mm. Hg. An electrocardiogram showed normal sinus rhythm, left ventricular hypertrophy, and diaphragmatic myocardial infarction with peri-infarction conduction defects. Conventional chest roentgenograms showed multiple calcifications of the apex of the heart extending below the diaphragm (Fig. 6, *A*, *B* and *C*). The preliminary circulation time (decholin) was prolonged (15 seconds). Angiocardiography showed an irregular aneurysm of the left ventricle which was surrounded superiorly by thrombus. Inferiorly, below the apex of the heart, considerable thrombus and calcifications were demonstrated (Fig. 6, *D*, *E* and *F*).

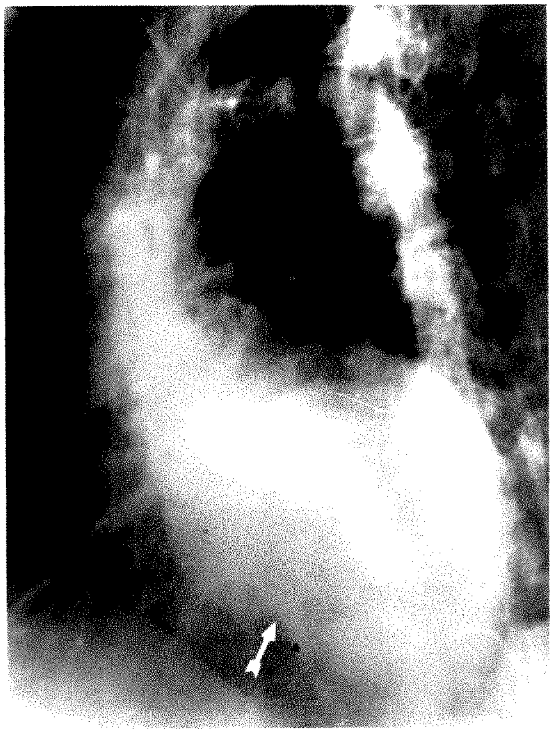

Fig. 5. Case v. Angiocardiogram showing another huge apical myocardial aneurysm of the inflow portion of the left ventricle (arrow).

Case vii. *Asymptomatic ventricular aneurysm.* A 62 year old Caucasian man (N.Y.H. No. 935 304) was admitted on December 12, 1962, complaining of fever, cough, and right chest pain of 12 days' duration. He had had pneumonia in 1931 and 1951. He recalled retrosternal pressure and pain radiating to both shoulders and lasting 12 to 18 hours after chopping wood in 1962. He had consulted his physician who found elevation of the serum transaminase and depressed ST segments in the electrocardiogram. The patient had insisted on going on vacation and did not return until the present admission. On examination, he had mild fever, respiratory distress, dullness, rales, and a pleural friction rub at the right base of the lung. The heart was regular and the blood pressure was 130/80 mm. Hg. A systolic (Grade 1) murmur was heard over the apex. The venous pressure was normal. The white blood cell count was elevated (11,200/mm.^{3}), the sputum culture yielded a heavy growth of

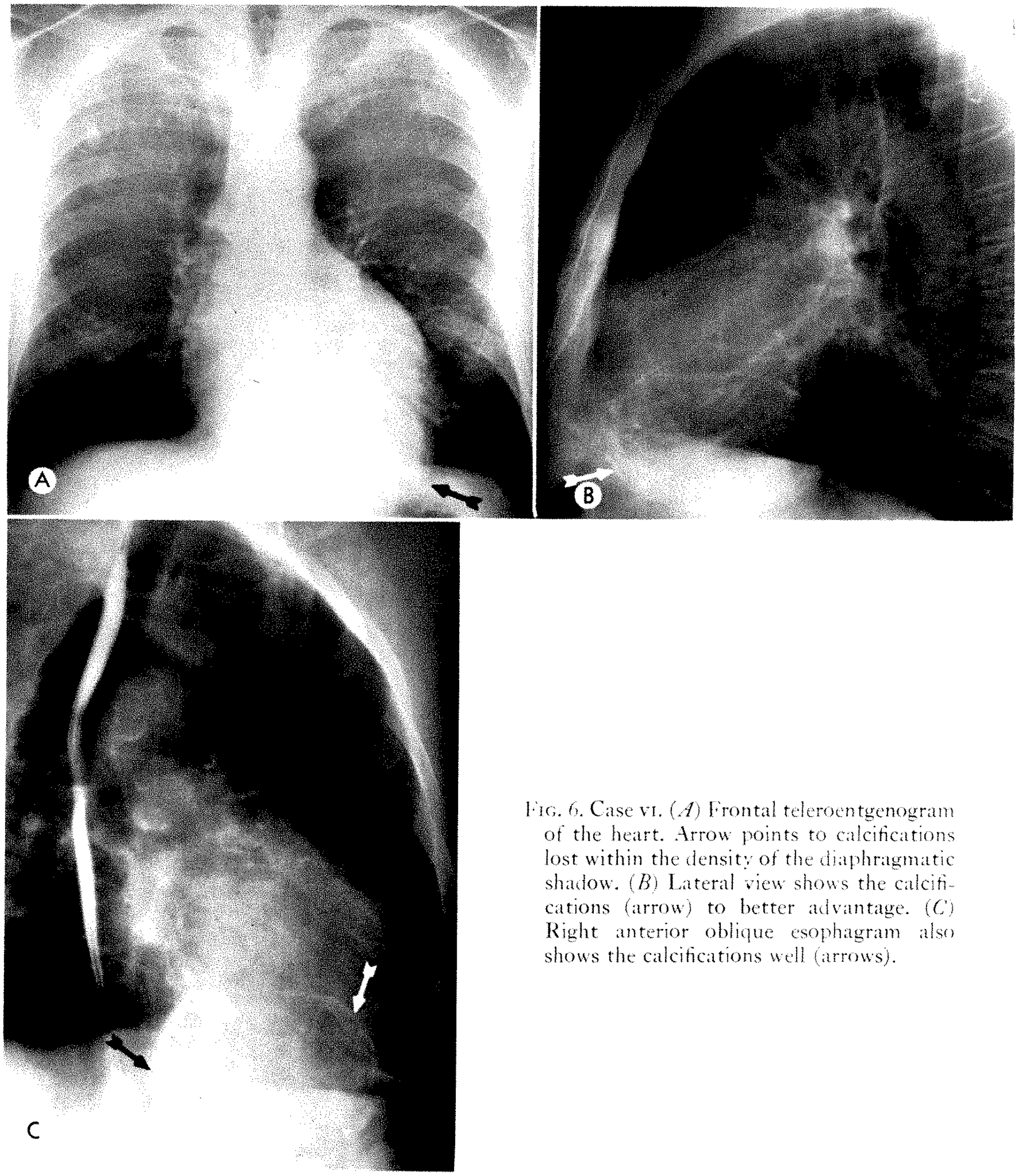

Fig. 6. Case vi. (*A*) Frontal teleroentgenogram of the heart. Arrow points to calcifications lost within the density of the diaphragmatic shadow. (*B*) Lateral view shows the calcifications (arrow) to better advantage. (*C*) Right anterior oblique esophagram also shows the calcifications well (arrows).

pneumococcus, and the sedimentation rate was 35. Roentgenograms of the chest revealed pneumonia at the right base. The heart was enlarged and contained a nonpulsatile bulge along the posterior wall of the left ventricle (Fig. 7, *A-D*). Electrocardiographic studies showed normal sinus rhythm, occasional premature contractions, and anterolateral myocardial infarction. After the pneumonia subsided, intravenous angiocardiographic studies on February 20, 1963, revealed an aneurysm jutting from the posterior border of the heart which measured 4 by 5 cm. during diastole and was surrounded by a thrombus measuring 2 to 3 cm. in thickness (Fig. 7, *E* and *F*). Cineangiocardiography revealed vigorous pulsations of the inflow portion of the left ventricle, but contractions within the aneurysm were stagnant, sluggish, and feeble. The patient was treated with antibiotics and discharged on

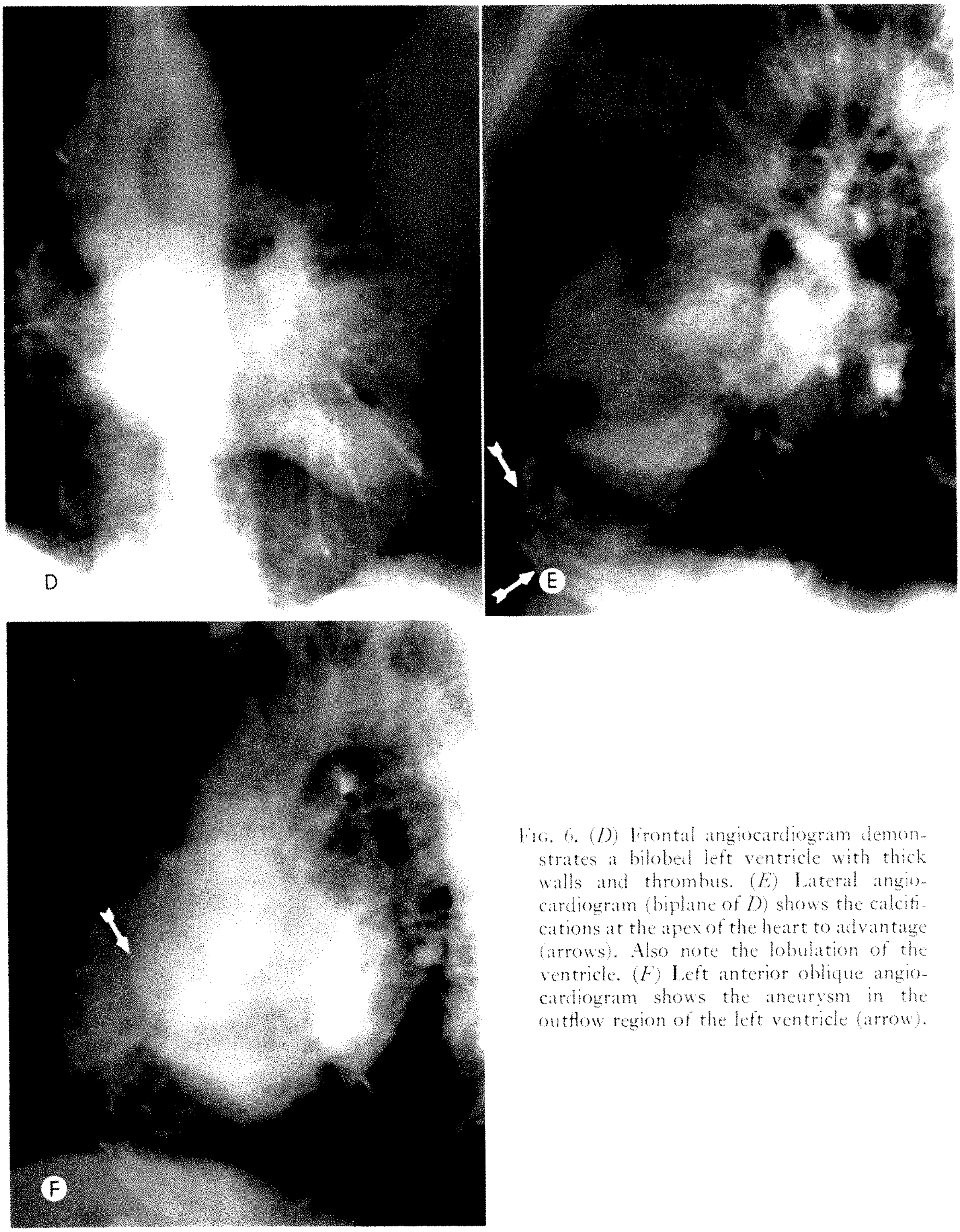

Fig. 6. (*D*) Frontal angiocardiogram demonstrates a bilobed left ventricle with thick walls and thrombus. (*E*) Lateral angiocardiogram (biplane of *D*) shows the calcifications at the apex of the heart to advantage (arrows). Also note the lobulation of the ventricle. (*F*) Left anterior oblique angiocardiogram shows the aneurysm in the outflow region of the left ventricle (arrow).

February 27, 1963. He has been maintained on digitalis and has remained asymptomatic.

Case viii. *Ventricular septal aneurysm.* A 62 year old Caucasian man (N.Y.H. No. 917 638) was admitted on July 11, 1963, complaining of cough and dyspnea of 1 year's duration. He gave a history of recurrent attacks of dyspnea and chest pain, the last one 3 days prior to admission. On examination, the neck veins were distended, the liver was enlarged 6 cm. below the right costal margin, and moist rales were

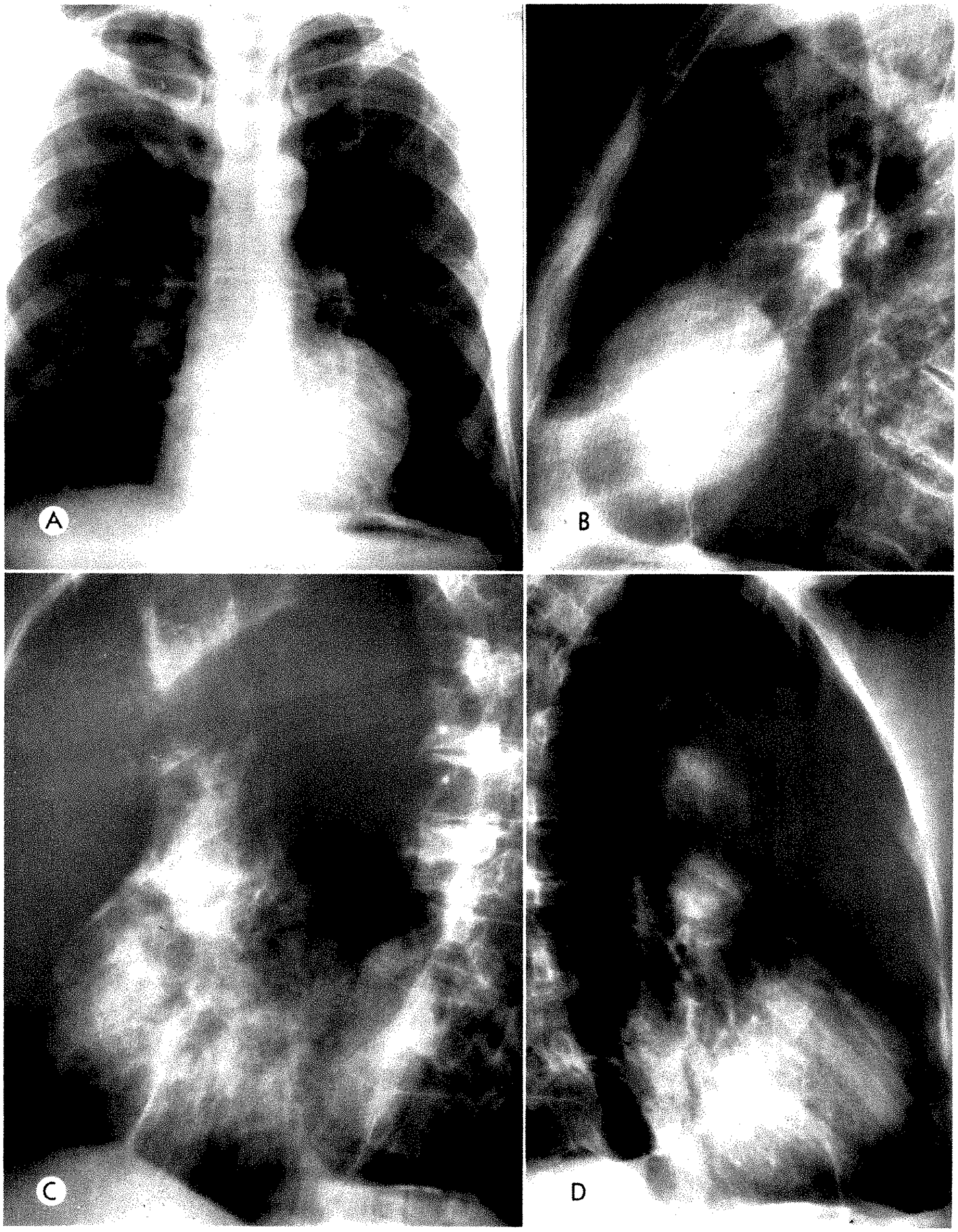

FIG. 7. Case VII. (*A*) Frontal teleroentgenogram of the chest showing unusual rounding of the contour of the left ventricle. (*B*) Lateral view showing a rounded mass in the center of the cardiac silhouette. (*C*) The left anterior oblique view showing the mass to be behind the heart. (*D*) In the right anterior oblique view, the ventricular aneurysm is not discernible.

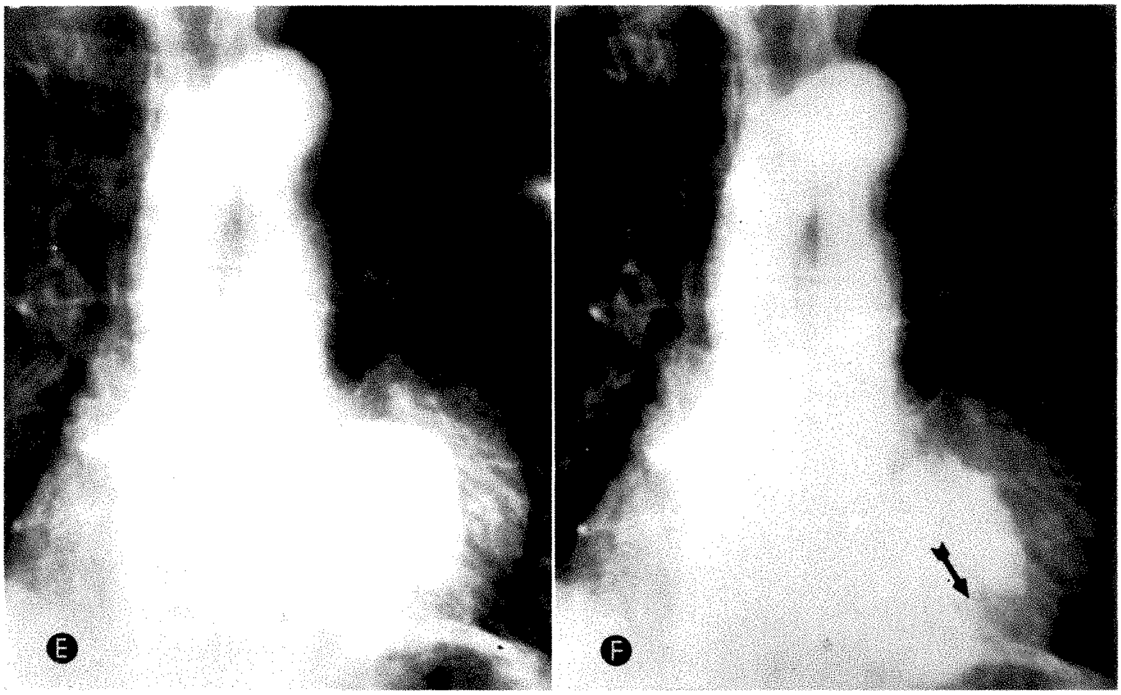

Fig. 7. (*E*) Frontal angiocardiogram, during diastole, shows an aneurysm of the left ventricle surrounded by thick wall and thrombus. (*F*) Frontal angiocardiogram, in systole, reveals the neck of the aneurysm (arrow).

heard at the bases of the lungs. The heart was enlarged, there were no murmurs, and the blood pressure was 110/70 mm. Hg. The electrocardiogram showed regular sinus rhythm, rate 90, marked left axis deviation, and old diaphragmatic and anterior wall myocardial infarctions. The roentgenogram of the chest showed moderate cardiac enlargement without bulging of the contours. Angiocardiograms on August 1, 1963, showed a 3 cm. in diameter aneurysm of the ventricular septum (Fig. 8, *A* and *B*). Anticoagulant therapy was prescribed and the patient was discharged. He has attended the outpatient department since then and was well on his last visit on June 30, 1965.

Case ix. *Pericardial effusion complicating anterolateral myocardial aneurysm.* (Presented through the courtesy of Dr. Barry T. Held of The Roosevelt Hospital, New York City.) A 64 year old Caucasian man developed severe chest pain and dyspnea in October, 1964. The electrocardiogram showed anterolateral myocardial infarction and a bulge was visible in the apicolateral region of the cardiac silhouette (Fig. 9, *A* and *B*). Frontal intravenous angiocardiograms showed an unsuspected pericardial effusion surrounding and elevating the heart, and a huge left ventricular aneurysm (Fig. 9, *C* and *D*). He improved after diuretic therapy; surgical treatment was not advised. He has remained well and has been free of dyspnea and chest pain.

Case x. *Operation not advised for patient with huge anterolateral myocardial aneurysm.* A 52 year old Caucasian man (N.Y.H. No. 1008703) was admitted on March 16, 1963, with a history of 2 attacks of myocardial infarction, 4 and 2½ years previously. The last attack was associated with aphasia, and right arm and leg weaknesses which cleared completely after a few days. Six weeks prior to admission, sudden onset of dyspnea occurred while driving a car, and he was rushed to another hospital where electrocardiographic studies failed to reveal an acute myocardial infarction. Severe bulging of the left ventricle was noted and he was transferred to this hospital.

Physical examination revealed a moderately obese man in no distress. The lungs were clear. The heart was enlarged, there were no murmurs, and the rate was regular. The blood pressure was 140/90 mm. Hg. The electro-

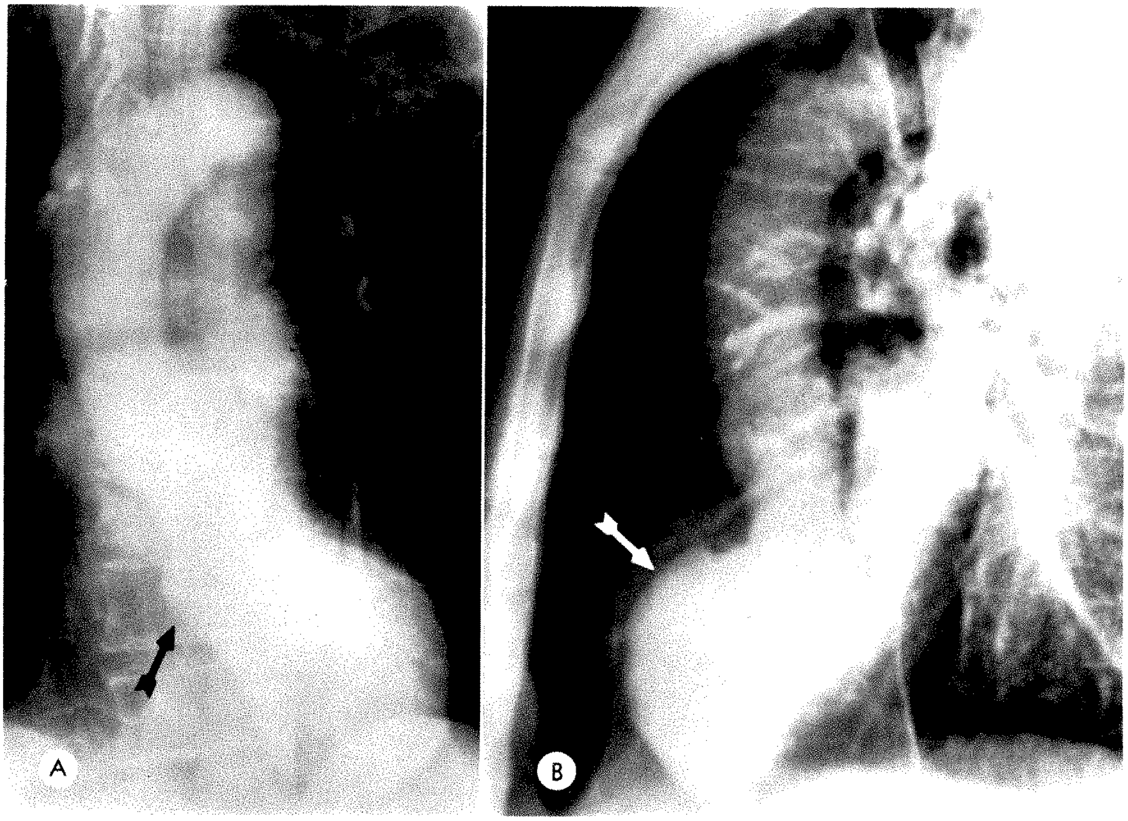

Fig. 8. Case viii. (*A*) Frontal angiocardiogram showing a ventricular septal aneurysm (arrow). (*B*) Lateral angiocardiogram (biplane of *A*) also showing the septal aneurysm (arrow).

cardiogram showed marked left axis deviation, anterolateral myocardial infarction of long standing, marked ST segment abnormalities, and peri-infarctional conduction defects consistent with a ventricular aneurysm. Roentgenographic studies of the heart showed a huge left ventricular aneurysm (Fig. 10, *A*, *B* and *C*) which was devoid of pulsations. The preliminary circulation time (decholin) was prolonged (17 seconds). Biplane intravenous angiocardiograms on March 16, 1965 showed a large saccular aneurysm, measuring 8.5 by 10 cm., located at the apex of the heart and surrounded by thrombus measuring 3 cm. in diameter (Fig. 10, *D* and *E*). Cineangiocardiography revealed vigorous contractions of the inflow portion of the left ventricle with blood flow into a huge flaccid and rarely contracting aneurysm. Surgical excision of the aneurysm was not advised because of the many attacks of myocardial infarction. It was also feared that excision of the aneurysm would leave too little cardiac chamber for proper hemodynamic action of the ventricle.

Case xi. *Huge posterolateral ventricular aneurysm following lobectomy for tuberculosis.* A 45 year old Caucasian woman (N.Y.H. No. 913 927) was admitted on June 9, 1962, for right upper and middle lobe lobectomies because of active pulmonary tuberculosis. Cough, expectoration, fever, and positive sputum had persisted for many months. Operation on August 30, 1962, was uneventful, but 48 hours later the patient became severely dyspneic and developed atrial fibrillation. The blood transaminase was elevated although after digitalis therapy regular sinus rhythm was restored. In a period of 10 weeks, the contour of the heart changed radically. A large bulge along the upper outer contour of the left ventricle became evident, whereas prior to operation the heart was normal in size. Because of the ventricular aneurysm, anticoagulant therapy was begun. Congestive heart failure complicated the convalescence and delayed discharge until January 26, 1963. The sputum cultures became negative 4 months prior to discharge.

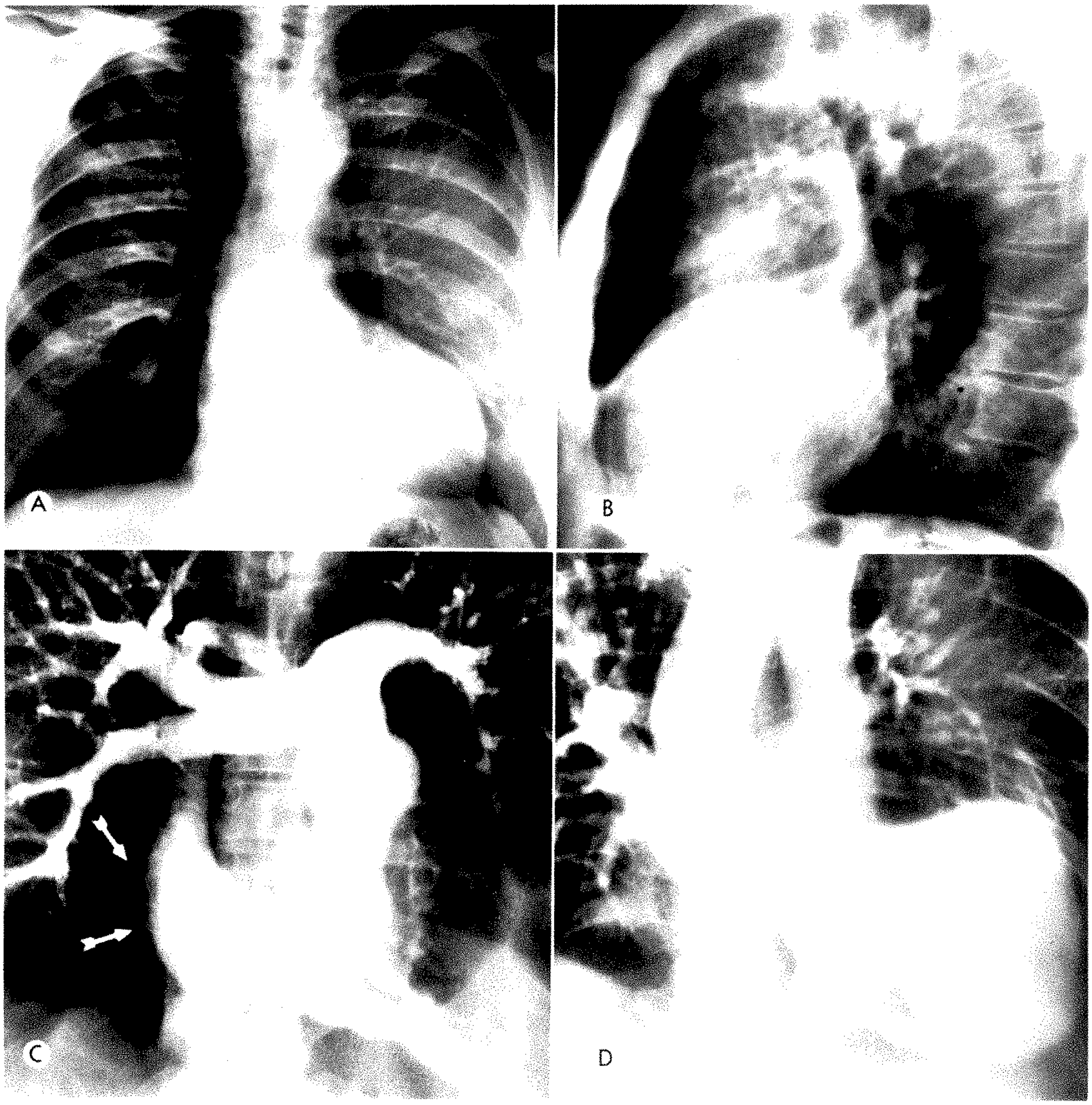

Fig. 9. Case IX. (*A*) Frontal teleroentgenogram of the chest showing enlargement of the left ventricle. (*B*) Lateral view showing a centrally rounded dense mass within the cardiac shadow. (*C*) Frontal angiocardiogram shows a pericardial effusion extending beyond the opacified outer wall of the right atrium (arrows) and elevating the heart above the diaphragm. (*D*) Frontal angiocardiogram shows the huge ventricular aneurysm occupying the entire outflow portion of the left ventricle.

The patient attended the cardiac and anticoagulant clinics and was well until March 24, 1963, when palpitations and atrial flutter, rate 180 with alternating 2:1 and 4:1 block, occurred. Four hours later there was spontaneous restoration of regular sinus rhythm, rate 96. The electrocardiogram showed left ventricular hypertrophy and ST and T wave abnormalities consistent with an old anterolateral myocardial infarction. She was discharged on March 29, 1963, and was referred for angiocardiography on April 8, 1965. The blood pressure was 110/74 mm. Hg, the venous pressure was slightly elevated, and the modified circulation time (decholin) was 16 seconds. The preliminary roentgenogram of the chest showed marked cardiac enlargement with a huge bulge along the upper border of the left cardiac silhouette (Fig. 11*A*). Biplane angiocardiography revealed a huge ventricular aneurysm, measuring 10 by 10.5 cm., in the posterolateral wall of the left ventricle (Fig. 11, *B* and *C*).

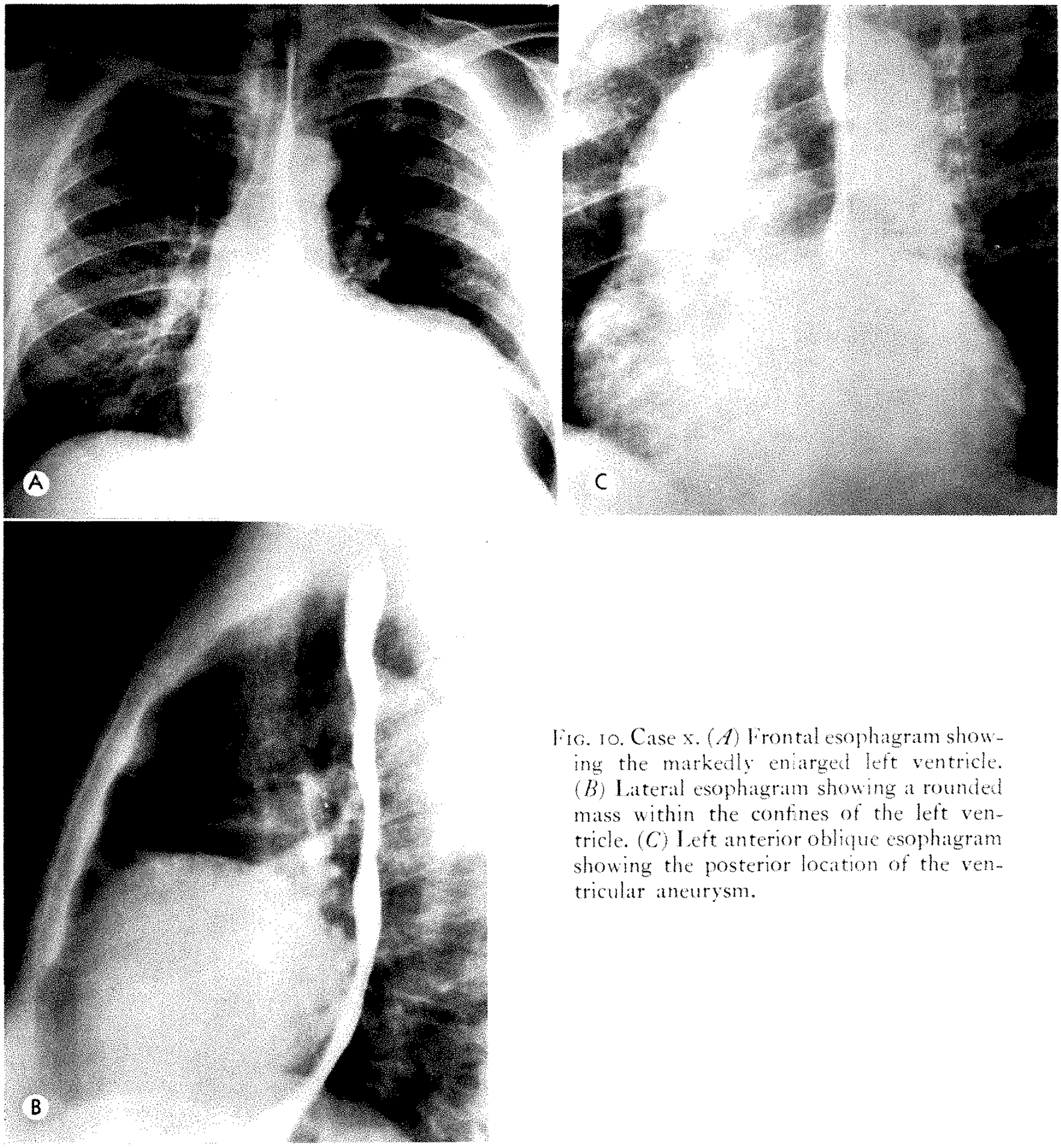

FIG. 10. Case X. (*A*) Frontal esophagram showing the markedly enlarged left ventricle. (*B*) Lateral esophagram showing a rounded mass within the confines of the left ventricle. (*C*) Left anterior oblique esophagram showing the posterior location of the ventricular aneurysm.

FINDINGS

Eight of the 11 patients with ventricular aneurysm were dyspneic on exertion; the extent varying with the degree of heart failure. Fifty per cent of the patients also had coronary disease insufficiency evidenced by angina. Two patients had cardiac arrhythmia—atrial flutter and fibrillation (Cases VI and XI). One patient was asymtomatic after recovery from pneumonia (Case VII). The electrocardiograms of all the patients showed either anterolateral or antero-apical myocardial infarctions. All but 3 of the patients had anterolateral or antero-apical ventricular aneurysms. One patient had a ventricular septal aneurysm (Case VIII, Fig. 8, *A* and *B*), and 2 others had aneurysms involving the postero-apical portion of the ventricle (Cases VII and XI). Linear calcifications along the thinned lateral wall of the aneurysm were found in 3 cases (Fig. 1; 2, *A* and *B;* and

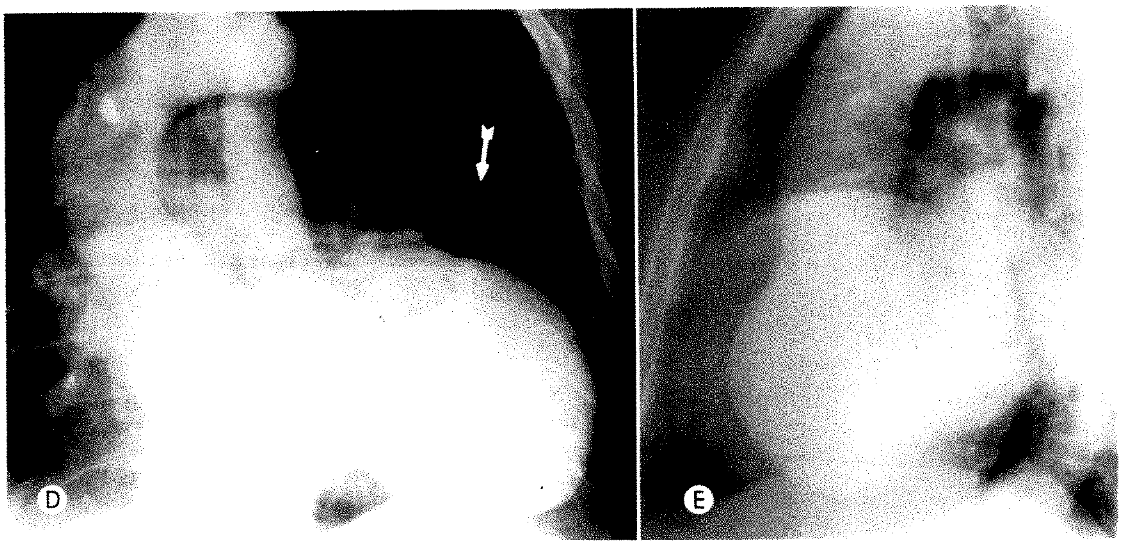

Fig. 10. (*D*) Frontal angiocardiogram shows the huge apicolateral and posterior position of the aneurysm surrounded by thick thrombus (arrow). (*E*) Lateral angiocardiogram (biplane of *D*) shows the central location of the ventricular aneurysm.

3*A*). Massive calcifications of the diaphragmatic border of the left ventricle were seen in another patient (Case IV, Fig. 6, *A-F*). Right heart catheterization was performed in 2 patients (Cases IV and VI). In none of these were the hemodynamic disturbances sufficient to warrant surgical excision of the aneurysm. Another patient (Case V) was explored and aneurysmectomy was not considered warranted—this decision was made prior to the advent of present day open-heart techniques. Surgery was not offered 1 patient because he did not have complaints relative to the aneurysm (Case VIII). In 2 others, mild symptoms contraindicated surgery (Cases VII and IX). One patient was denied aneurysmectomy because of multiple myocardial infarctions (Case X) and another because of previous lobectomies for treatment of tuberculosis (Case XI).

DISCUSSION

Holmes and MacFadyen[12] recently published 6 cases of postinfarction ventricular aneurysms. They studied 5 patients with selective left ventricular angiography and thoroughly reviewed the literature on the subject. The clinical diagnosis of ventricular aneurysm depends upon the recognition of an unusual bulge of the cardiac silhouette. This can best be seen along the apical and lateral surfaces of the left ventricle. If the bulge is small or indistinct, it tends to blend with the contours of the enlarged left ventricle, especially below the left diaphragm, and escapes recognition. Since the ventricular septum is within the cardiac silhouette, it is, therefore, inaccessible. Paradoxical motion of the aneurysm is not a reliable sign because it may be present in patients who have suffered myocardial infarctions without aneurysmal formation,[15] and, rarely, it may be a normal finding.[14] For these reasons, the pathologic diagnoses of postinfarctional myocardial aneurysms are made more often than the clinical.[1,8,9,20]

Some authors[22] have objected to the term of ventricular aneurysm, stating that the name of aneurysm should be reserved for arteries, since an aneurysm usually involves vessels, especially arteries. However, usage of the designation of ventricular aneurysm for a localized bulge of the ventricle has become generally accepted and firmly established in the literature. Only ventricular aneurysms following arteriosclerotic myocardial infarctions are herein

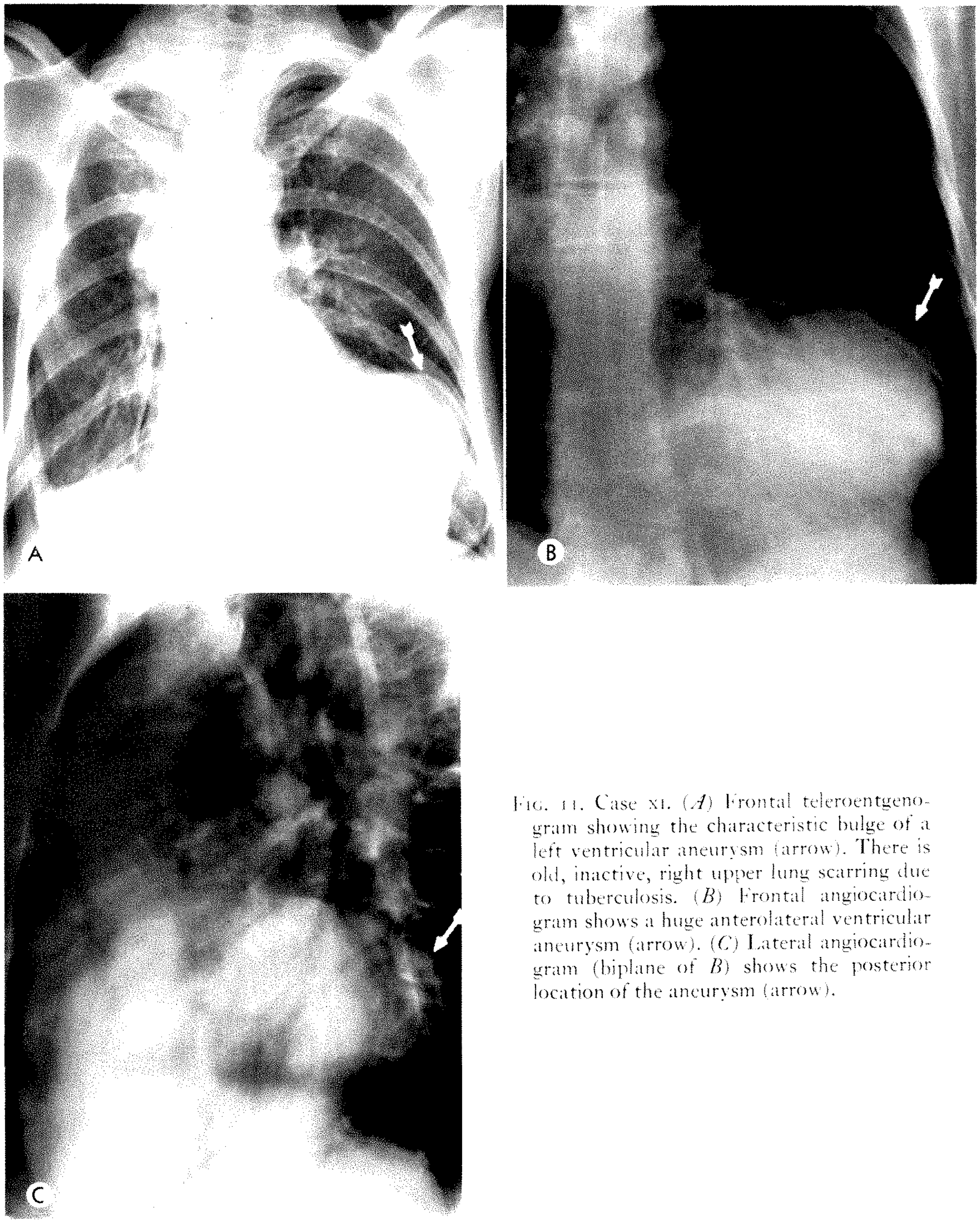

Fig. 11. Case xi. (*A*) Frontal teleroentgenogram showing the characteristic bulge of a left ventricular aneurysm (arrow). There is old, inactive, right upper lung scarring due to tuberculosis. (*B*) Frontal angiocardiogram shows a huge anterolateral ventricular aneurysm (arrow). (*C*) Lateral angiocardiogram (biplane of *B*) shows the posterior location of the aneurysm (arrow).

discussed. Other causes of left ventricular aneurysms such as trauma (stab wounds and postoperative ventricular pseudo-aneurysms[23]), infections (gumma, acute endocarditis, tuberculosis, and echinococcus), and congenital (anomalous origin of left coronary artery from the pulmonary artery[16,24]) are not included.

Selective cine coronary arteriography[10] and coronary arteriography[12] have shown that total occlusion of the anterior descending branch of the left coronary artery is usually responsible for development of a left ventricular aneurysm. If the myocardial necrosis is limited to the apical and anterolateral portions of the left ventricle and if the other coronary arteries are healthy and permit sufficient collateralization with the occluded coronary artery, necrosis, fibrous healing, and aneurysmal formation may follow in short order[9,21] and cause few symptoms. Aneurysms involving the posterior portion of the left ventricle, however, are prone to involve the papillary muscles, produce mitral insufficiency, and arrhythmia.[10] Only 1 patient in this series was asymptomatic, the others suffered from angina, arrhythmia, and congestive heart failure. Because of these complications and even though the mortality for excision of the aneurysm is high (up to 25 per cent), aneurysmectomy is advocated by many.[2–5,10–12,18] Cardiac catheterization, particularly left ventricular pressure determinations, is important for selection of the case suitable for operation.[5,10,11,18] Selective coronary arteriography is especially valuable for assessment of the state of the remainder of coronary circulation, since severe arteriosclerotic disease of the right coronary and circumflex coronary branch of the left coronary arteries may contraindicate operation.[10]

Enlargement of the heart with a localized bulge of the left ventricle in the conventional roentgenogram of the chest, particularly following myocardial infarction, is suggestive of ventricular aneurysm. When the bulge contains linear calcification (Fig. 2*A* and 3*A*) and pulsates vigorously, paradoxically, or not at all, the diagnosis is certain. Sometimes, the heart may be minimally enlarged (Fig. 7*A*) or contain massive calcifications (Fig. 6, *B* and *C*). In such instances, roentgenography and roentgenoscopy in various positions are necessary to fully visualize the aneurysm (Fig. 7, *B*, *C* and *D*; 9*B*; and 10, *B* and *C*) as well as the full extent of the calcifications. Also, the anterolateral and apical portions of the left ventricle, which ordinarily blend with the diaphragm, need to be thoroughly studied by slightly turning the patient to the right anterior oblique position (Fig. 2*A* and 6*C*).

Contrast visualization of the left ventricle, either by intravenous or selective routes, further refines the diagnosis of ventricular aneurysm. The selective left ventricular technique is particularly valuable because it enables the recording of pressures and the performance of coronary arteriography. Cineangiography employing contrast medium also enhances the study of the motion of the heart, particularly of the aneurysm.[10,12] This has also been achieved by cineangiocardiography of the cardiovascular system by the intravenous injection of contrast agent (Cases VI, VII, and X). Unusual thinning of the aneurysmal wall, and the size, shape, and amount of thrombus in it are regularly demonstrated (Fig. 1). Aneurysms of various sizes and shapes may be readily seen (Fig. 2, *B* and *C*; 3*B*; 4; 5; 6, *D*, *E*, and *F*; 7, *E* and *F*; 8, *A* and *B*; 9*D*; 10, *D* and *E*; and 11, *B* and *C*). Intravenous angiocardiography, by the serial visualization of the entire cardiovascular system, also permits recognition of disease of other parts of the heart, *i.e.*, an unsuspected pericardial effusion (Fig. 9*C*). The preliminary circulation time (decholin), when routinely used to establish the rate of blood flow for serial angiography,[27] is a reliable measure of left ventricular function and of the emptying time of the aneurysm. Further hemodynamic data regarding the ventricle and aneurysm may also be obtained by the simultaneous electrocardiographic recording of the cardiac cycle. Obstruction and delay of blood flow through the aneurysm may be assessed thereby. Probably the greatest usefulness of angiocardiography is that it permits differentiation of ventricular aneurysms from other heart lesions such as cardiac tumors, adjacent mediastinal masses, pericardial cysts, and pericardial effusions.[26]

SUMMARY AND CONCLUSIONS

Intravenous angiocardiography in a series of 11 patients with arteriosclerotic (post-myocardial infarction) left ventricular aneurysms permitted evaluation of the size and shape of the aneurysm, thickness of the myocardial wall, and thrombus. Assessment of the hemodynamic effect of the aneurysm was also obtained from study of the emptying and contraction states of the opacified aneurysm and ventricle. Conventional roentgenography and roentgenoscopy in all positions, by demonstrating unusual bulging, calcification, and pulsation of the ventricle, suggested the diagnosis of ventricular aneurysm in 10 cases; angiocardiography confirmed it. In 1 patient, a ventricular septal aneurysm, undetected by conventional roentgenographic techniques, was revealed by angiocardiography. Selective left ventricular angiography and coronary arteriography are particularly recommended for securing significant indications for operative treatment (aneurysmectomy) because these techniques permit both the recording of pressures in the ventricle and aneurysm and the evaluation of the state of the coronary arterial circulation. Finally, angiocardiography allows differentiation of myocardial aneurysms from cardiac tumors, adjacent mediastinal tumors, and pericardial cysts and effusions.

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Since this paper was submitted, 3 new publications on the physiologic,[1] electrocardiographic,[2] and surgical treatment[3] of postinfarction left ventricular aneurysms have appeared:

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ROENTGEN DIAGNOSIS OF LEFT VENTRICULAR ANEURYSM*

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THE successful surgical treatment of left ventricular aneurysms as reported by Björk,[2] Cooley *et al.*[3] and others has emphasized the need for a method by which the exact diagnosis of this condition can be made. Clinically, the diagnosis is difficult. Abrams, Edelist, Luria and Miller in 1963 found only 4 of 65 autopsy cases in which the correct diagnosis was made ***intra vitam***. Although in 1922 Sézary and Alibert[6] reported the first roentgen diagnosis of left ventricular aneurysm, the diagnosis is not readily made from conventional chest roentgenograms and fluoroscopy.[7] The findings at kymography and electrokymography are often difficult to interpret.[1] Cineroentgenographic studies of the contours of the heart for the purpose of arriving at a diagnosis of left ventricular aneurysm are not only difficult but may be misleading.[5] Angiocardiography has been employed by Dotter and Steinberg[4] and others; however its use has been limited in the series of left ventricular aneurysms reported. Furthermore, since there are no comparative studies on the diagnostic value of the conventional roentgen examination, it was felt that a report of our experience with 14 operated on cases of left ventricular aneurysm would be of interest.

MATERIAL

The patients were all men and their ages varied from 22 to 60 years. All patients had conventional chest examinations before surgery, including fluoroscopy and cinerecording of the movements of the left ventricular wall. Angiocardiography, using the full size biplane technique, was performed in all patients. In 11 cases cineangiocardiography in two different projections was also performed. In addition, coronary angiograms were obtained in 8 of the patients.

Table I

FINDINGS AT CONVENTIONAL ROENTGEN EXAMINATION OF THE CHEST IN 14 CASES OF LEFT VENTRICULAR ANEURYSMS

Enlargement of the heart	10
Left ventricular enlargement	8
Abnormal left ventricular contour	6
Calcifications in left ventricular wall	1
Abnormal (decreased) motility of part of left ventricular wall	7

The positive findings at conventional roentgen examination of the heart are shown in Table 1. Only 6 of the 14 cases showed a definite abnormal contour of the left ventricle with a more or less localized bulge. In these cases an abnormal motility of this part of the left ventricular wall could be seen on fluoroscopy and on cineroentgenography in different projections. The frequently described paradoxical movements of this part of the left ventricular wall were not seen in these cases. Tracings from the cineroentgenograms showed that in 7 cases there was definitely decreased motility of part of the left ventricular wall. In some cases an apparent outward bulging of this part was thought to be observed at fluoroscopy; however, the tracings from the cineroentgenograms showed this to be an observer's error caused by the aneurysmatic part of the ventricular wall being immobile while the rest of the ventricular contour contracted. Thus, in less than half of these selected cases of left ventricular aneurysm did the conventional heart examination give evidence of an aneurysm. In 4 cases, no cardiac abnormalities whatsoever could be detected and in the remaining 4 moderate enlargement of the heart and/or the left ventricle could be detected.

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The findings at angiocardiography are shown in Table II. Definite left ventricular enlargement was present in 8 of the 14 cases; 13 cases had increased residual blood in the left ventricle at end systole. The only case not showing this was one of a peculiar, probably congenital, aneurysm with a very narrow communication with the left ventricle. A true aneurysm, *i.e.*, an outpouching of part of the ventricular wall, was seen in 9 cases. In the remaining 4

TABLE II

FINDINGS AT ANGIOCARDIOGRAPHY IN 14 CASES OF LEFT VENTRICULAR ANEURYSMS

Left ventricular enlargement	8
Increased residual blood in left ventricle	13
Left atrial enlargement	3
Mitral insufficiency	2
Local left ventricular aneurysm	9
Stiff, smooth left ventricular wall	13
Paradoxical expansion of aneurysm	2
Coronary angiogram positive (made in only 8 cases)	8

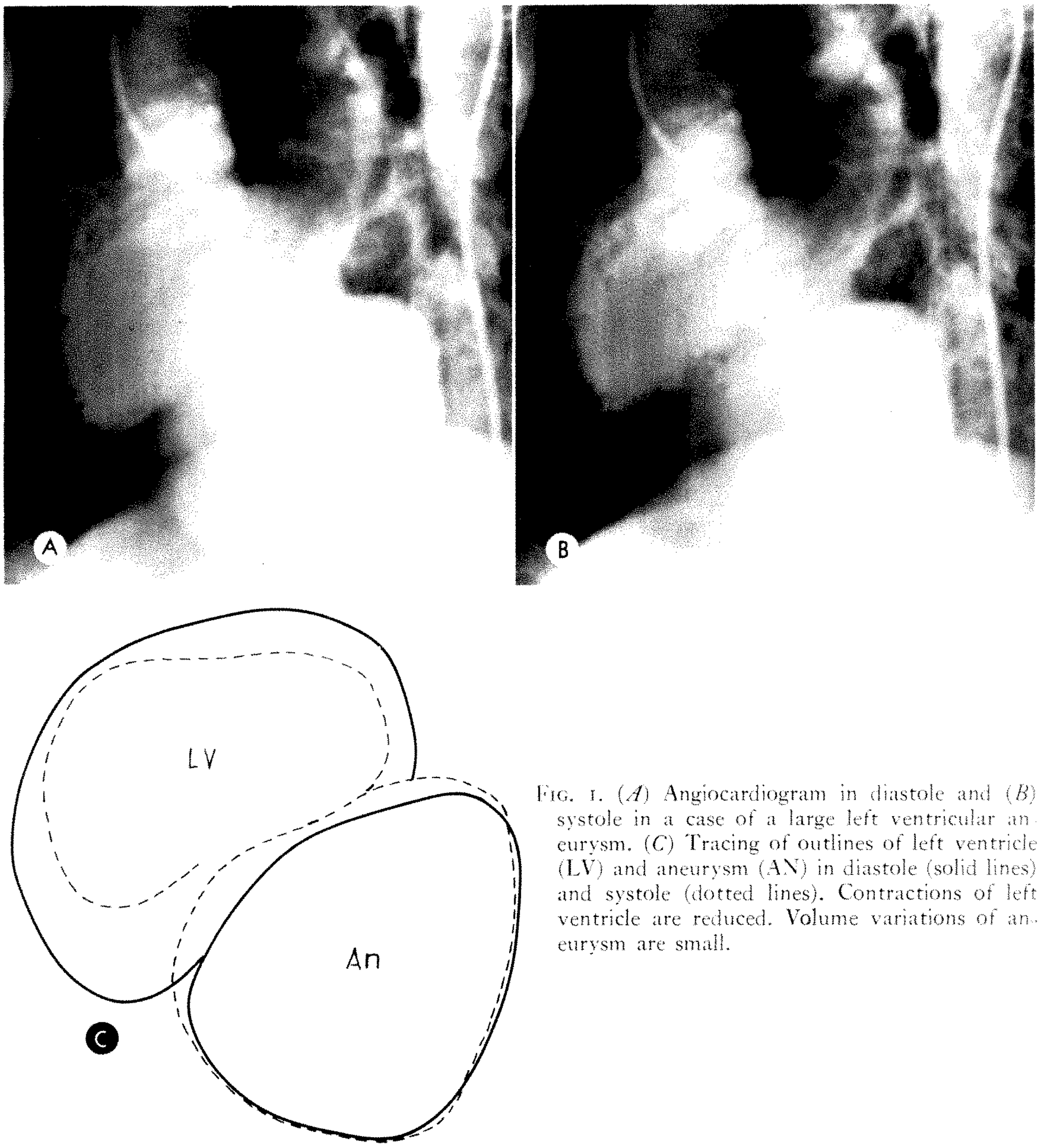

FIG. 1. (*A*) Angiocardiogram in diastole and (*B*) systole in a case of a large left ventricular aneurysm. (*C*) Tracing of outlines of left ventricle (LV) and aneurysm (AN) in diastole (solid lines) and systole (dotted lines). Contractions of left ventricle are reduced. Volume variations of aneurysm are small.

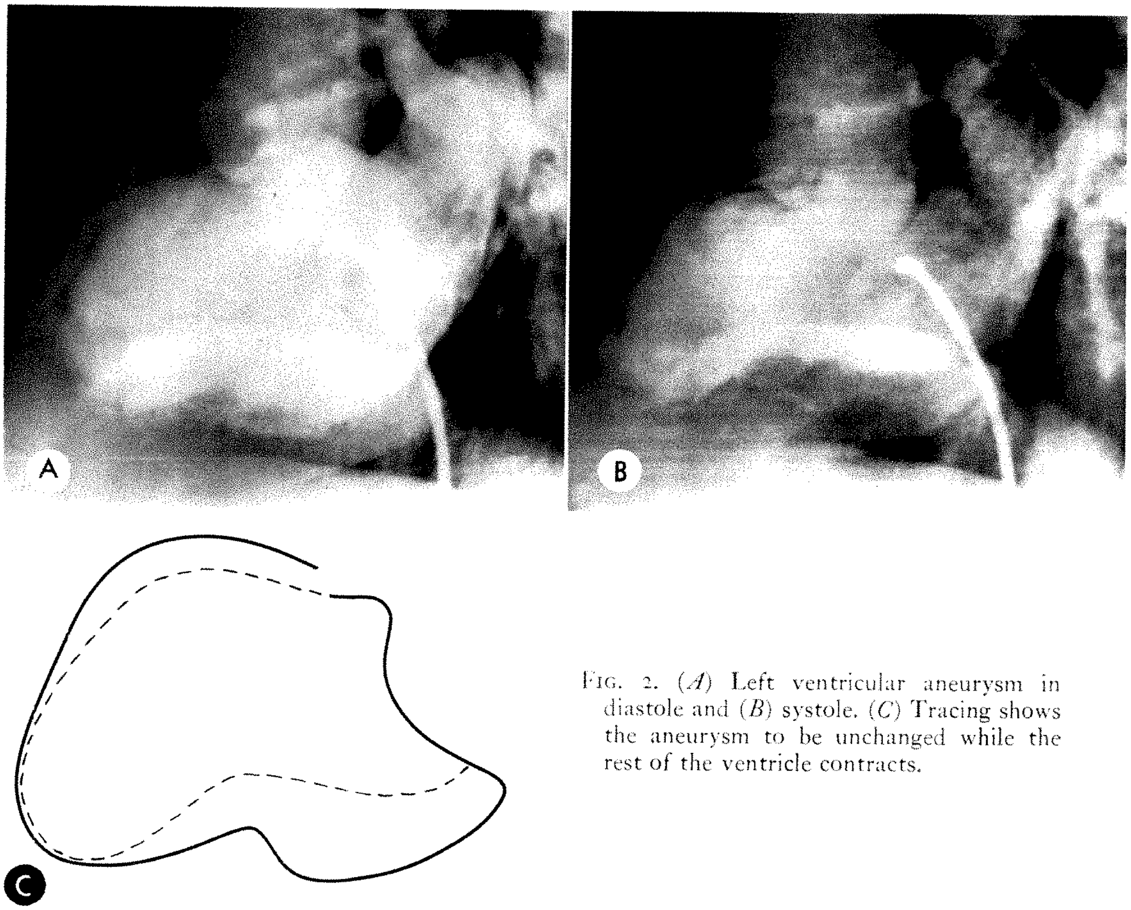

FIG. 2. (*A*) Left ventricular aneurysm in diastole and (*B*) systole. (*C*) Tracing shows the aneurysm to be unchanged while the rest of the ventricle contracts.

cases, there was an immobile smooth area on the left ventricular wall but no outpouching. The much described paradoxical expansion of the aneurysm in systole was seen only in 2 cases with very large aneurysms (Fig. 1, *A*, *B* and *C*). Even in these cases, this paradoxical expansion was slight. In most of the cases, the aneurysmatic part of the ventricle remained immobile during systole, while the remaining part of the ventricular wall contracted (Fig. 2, *A*, *B* and *C*). Mitral insufficiency was seen in 2 cases, probably due to interference with the function of the papillary muscles from the aneurysm. In 8 cases where coronary angiograms were obtained, occlusion of the main branches of the coronary arteries leading to the area of the aneurysm could be visualized. Cineangiocardiography was found to be very useful in demonstrating the immobile part of the left ventricular wall, consistent with ventricular aneurysm; however, it is important to chose the proper projection for this study, as shown in Figure 3, *A* and *B*. A difference in rotation of only 15 degrees may obscure this characteristic appearance of part of the left ventricular wall.

DISCUSSION

Even in this selected material, the roentgen diagnosis of left ventricular aneurysm could be made in only 6 of 14 cases at conventional chest examination. However, left ventricular angiocardiography proved most rewarding. The diagnosis was made in all cases except 1 in which a very small communication was present between the aneurysm and the left ventricle. The other 13 all had increased residual blood in left ventricle at end systole, indicating impairment of left ventricular function. The much

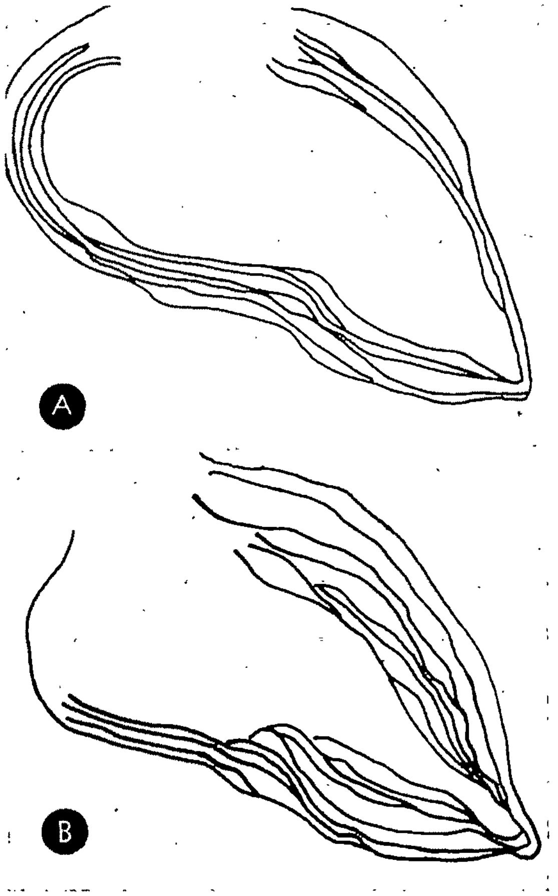

Fig. 3. Tracings from cine-angiocardiograms of left ventricular systole. (*A*) In the right anterior oblique projection, the aneurysmatic immobile part of the left ventricular wall is not seen. (*B*) By turning the patient 15° more, the aneurysm of the posterior wall is clearly demonstrated.

described paradoxical systolic expansion of the aneurysm was not particularly evident in this study. It seems that this so-called paradoxical movement represents rather a standstill of the aneurysmatic part of the left ventricular wall while the rest of it is contracting. When looking at the heart from the outside, a false impression of expansion of the aneurysm is obtained. Since the wall of the aneurysm is composed mainly of fibrous scar tissue with very little elastic components, it is difficult to understand that expansion of the aneurysm may occur except to a very small degree.

It would seem rational to use left ventricular angiocardiography extensively in suspected cases of left ventricular aneurysm. This method is highly reliable in providing the exact diagnosis and also permits an evaluation of the function of the left ventricle.

SUMMARY

In a series of 14 cases of left ventricular aneurysm, findings consistent with the diagnosis were present in only 6 cases at conventional roentgen examination of the chest. Left ventricular angiocardiography provided the correct diagnosis as well as information on left ventricular function in 13 of the cases.

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CARBON DIOXIDE CINEANGIOCARDIOGRAPHY IN THE DIAGNOSIS OF PERICARDIAL DISEASE*

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THE limitations of the clinical examination, electrocardiography, plain film roentgenography and conventional fluoroscopy in the diagnosis of pericardial effusion are well known. Reliance upon pericardiocentesis to establish the diagnosis entails some risk to the patient and has the added disadvantage that failure to recover pericardial fluid is not reliable evidence of absence of effusion. Opaque contrast angiography has been employed in the diagnosis of pericardial disease.[2,13,14] This is a relatively cumbersome procedure which also carries the risk of any injection of contrast medium. Carbon dioxide, on the other hand, has been used for a number of years in many institutions with only one fatality reported[5] associated with its use. However, the technique of using one or two films,[7,8,9,11,15] although very simple, has resulted in occasional difficulties both in the timing of the film sequence as well as some difficulties in interpretation. Stauffer *et al.*[11] in 1960 stated that some of the difficulties in interpretation could be obviated by the use of cineroentgenography or serial techniques regularly used with opaque angiography.

Carbon dioxide is 20 times more soluble in plasma than air or oxygen. This property allows the transient formation of a gas-blood level in the right atrium when the patient lies on his left side. This is the basis for the procedure. The usefulness of placing the patient in this position was established by Oppenheimer *et al.*[6] in studies of the treatment of venous air embolism. In this position even though the upper portion of the right ventricle may contain gas, the outflow tract of the right ventricle and the trunk of the pulmonary artery are kept dependent and filled with blood, so that flow to the lungs and systemic circulation is maintained.

Right ventricular pressure determinations by right heart catheterization and electrocardiography showed no significant alteration in 2 patients who received 50 and 60 cc. of intracardiac carbon dioxide in the supine position.[11] In animal experiments, there was a brief rise of 5 to 10 volumes per cent in the whole blood carbon dioxide content and a maximum decrease of 1 to 2 volumes per cent of oxygen content following the rapid injection of 7.5 cc./kg. body weight of carbon dioxide[1] into the systemic veins. Injections of the same amounts into the left heart and systemic arteries of dogs showed that even this was well tolerated.[1] Carbon dioxide angiography is contraindicated in patients with impaired mechanisms of CO_2 excretion, such as far advanced pulmonary emphysema, in whom danger of invoking severe CO_2 narcosis may occur. A relative contraindication is present in the severely ill patient with marked orthopnea who might not be able to maintain the left lateral decubitus position for the required length of time for proper filming. This report is the analysis of 100 cineroentgenographic intracardiac carbon dioxide examinations performed in 90 patients (Table 1).

TECHNIQUE

All patients should have chest roentgenograms to determine whether a right pleural effusion is present, since right pleural fluid may overlie the heart when the patient lies on the left side and give an erroneous impression of an abnormally thickened right cardiac border. In patients with mobile

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Table I

	Band		Fluid Wave		Flattening		Asymmetry	
	−	+	−	+	−	+	−	+
No. of Examinations	32	68	45	55	94	6	80	20
No. of Patients	30	60	39	51	85	5	72	18

right pleural effusions, intracardiac carbon dioxide angiography is postponed until after thoracentesis; in those with loculated effusions until after resolution of the fluid collection.

The patient is placed on a stretcher lying on his left side in front of the upright fluoroscopic table. A narrow stretcher is used, so that the image intensifier can be centered over the right atrium. A tank of 100 per cent carbon dioxide is attached to a syringe through a three way stop cock. A No. 18 or No. 19 needle is placed in the antecubital vein of the left arm and the needle and the vein are kept patent by the slow infusion of isotonic saline solution. A 50 cc. syringe is filled with carbon dioxide and all the tubing connected to the syringe and tank is flushed 10 to 15 times. After clamping the intravenous infusion tubing, 50 cc. of carbon dioxide is injected over a 2 to 3 second interval. The cine-film is started just prior to the injection to record the flow of carbon dioxide through the superior vena cava into the right atrium. The cine-film is run for 15 to 30 seconds and then the heart is viewed intermittently fluoroscopically until the carbon dioxide bubble in the right atrium has been completely absorbed before the patient is returned to the supine position.

RESULTS

In evaluating the cine-films, the following criteria were used: (a) degree of pulsation of the right cardiac border; (b) width of the opaque band; (c) presence or absence of a fluid wave; (d) flattening of the opaque band; (e) asymmetry of the opaque band; (f) gas in other structures; and (g) spurious shadows.

DEFINITION OF TERMS

Degree of pulsation of the right cardiac border was evaluated by observing the pulsatile movement of the right cardiac border with the patient in the left lateral decubitus position and grading the amplitude of pulsations from 0 to 3 plus, with 0 representing absence of pulsation and 3 plus normal pulsation.

The opaque band refers to the composite shadow formed by the endocardium, the myocardium, the two layers of pericardium and the mediastinal pleura, which separates the radiolucency inside the right atrium produced by the carbon dioxide from the radiolucency of the right lung. The opaque band, allowing for magnification, is normally less than 5 mm. thick.

Fluid wave is defined as a changing thickness of the opaque band during the cineroentgenographic examination.

Flattening of the opaque band refers to the loss of the normal convex upper curvature of the carbon dioxide shadow in the right atrium.

Asymmetry of the opaque band refers to a constant variation in width of the thickened opaque band with either the cephalad or caudad portion of the band thicker than its central portion.

Gas in other structures refers to carbon dioxide outside the right atrium. These structures have included the superior vena cava, the right ventricle, pulmonary arteries, the left atrium, pulmonary veins, the inferior vena cava, and the hepatic veins.

Spurious gas shadows designate gas other than carbon dioxide in structures such as the main stem bronchus, pulmonary blebs, etc.

Table II

	Band / Pulsation	thin → thick			
		0	1+	2+	3+
absent → normal	3+	25	9	4	2
	2+	3	6	5	0
	1+	4	8	12	2
	0	0	4	7	9

PULSATIONS

An attempt was made to correlate the degree of pulsation with the thickness of the opaque band (Table II). In only approximately 50 per cent of the cases was there any relation between decreased pulsation and thickening of the opaque band. These findings support the views stated in the literature[10] that there may be normal pulsations with as much as a liter of pericardial fluid.

OPAQUE BAND THICKNESS

Table III

Abnormal Band	60 patients
Fluid Waves	51 patients
No Fluid Waves	9 patients

Band thickness was graded 0 to 3 plus (0=less than 5 mm.; 1+=6 to 10 mm.; 2+=11 to 20 mm.; 3+=greater than 21 mm.). In our series of 100 examinations, 68 showed a band thicker than 5 mm. Pericardial abnormalities were proven in 33 of the 68 by pericardiocentesis, surgery, or autopsy. In the remaining 35 cases, there was clinical evidence of pericardial disease.

Normal band width (Fig. 1, *A* and *B*), *i.e.*, less than 5 mm., was found in 32 cases and of these 7 cases went to autopsy and showed no evidence of fluid. In the remaining 25 cases, none was proven to have pericardial disease.

Since pericardial fluid accumulates anterior to the heart to a greater degree than laterally[13] and shows no significant redistribution with changes in body position,[4] it was difficult to quantitate the amount of fluid present by measuring the degree of band thickening.

FLUID WAVES

Fluid waves were present in 51 of 60 patients (Table III) who had abnormal opaque bands. Of these 23 were proven to have fluid by pericardiocentesis, surgery or

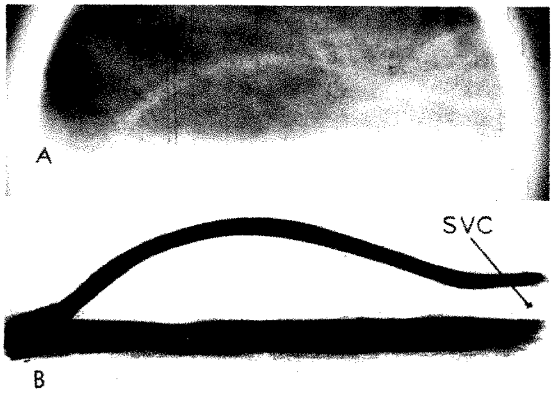

Fig. 1. (*A* and *B*) Normal atrial band. The band is less than 5 mm. in thickness allowing for magnification. SVC=superior vena cava.

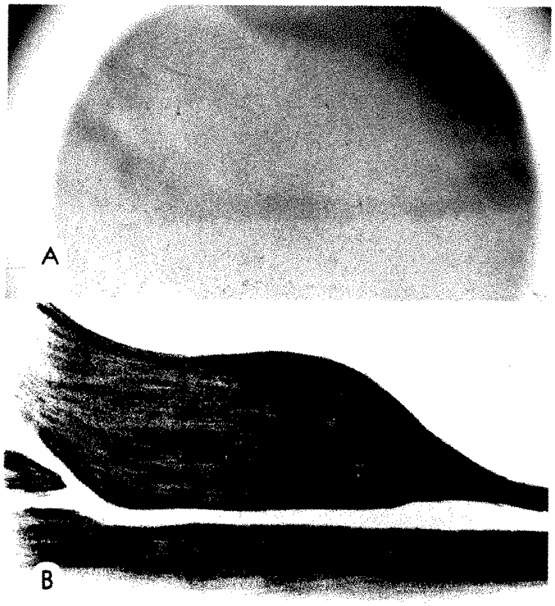

Fig. 2. (*A* and *B*) Flattening and asymmetry of band in patient who had constrictive pericarditis.

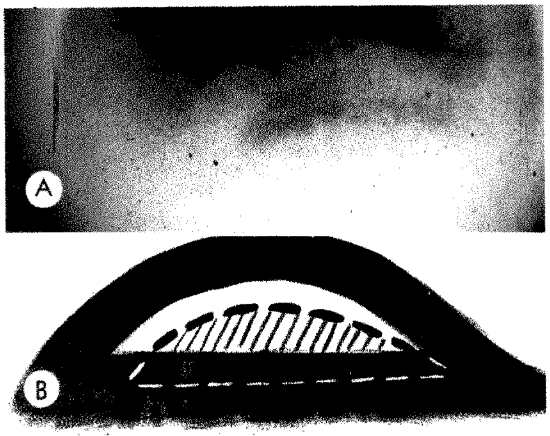

Fig. 3. (*A* and *B*) Symmetric band. Dotted outline represents fluid wave change with band remaining symmetric.

autopsy. Twenty-six patients were clinically diagnosed as having pericardial effusion and only 2 had clinical diagnoses of chronic pericarditis. No fluid waves were present in 9 of the 60 patients with abnormal opaque bands. In contrast, the clinical diagnoses in these patients were constrictive pericarditis in 2, traumatic hemopericardium in 2, pneumococcal chronic pericarditis in 1, idiopathic chronic pericarditis in 1, Hodgkin's infiltrative pericardium in 1, resolving myocarditis and pericarditis in 1, and uremic pericarditis in 1. Pericardial thickening was proven to be present in 6 of 9 patients without fluid waves.

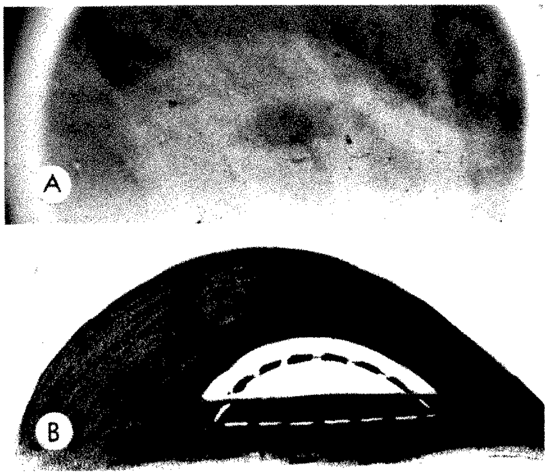

Fig. 4. (*A* and *B*) Asymmetric band. Dotted line represents fluid wave change with band remaining asymmetric.

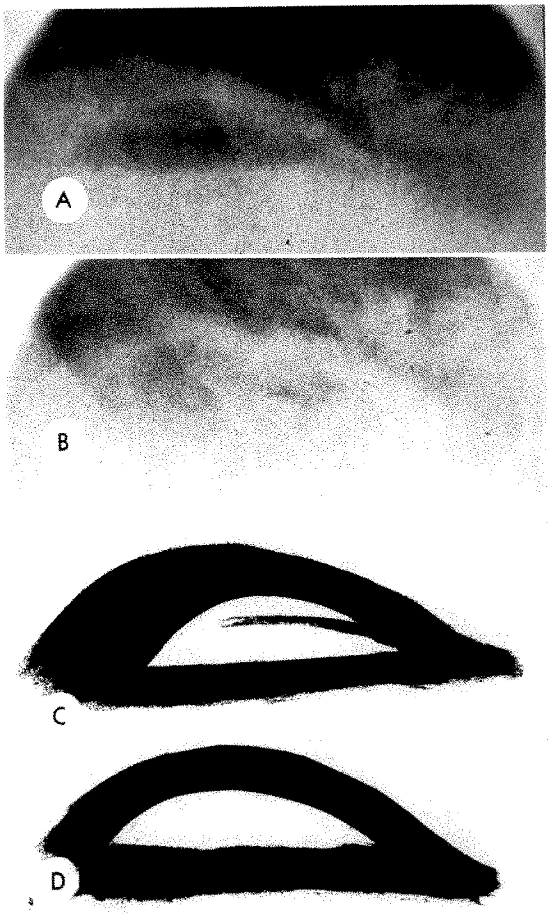

Fig. 5. (*A*–*D*) Changing symmetry. The thickened opaque band (*A* and *C*) changes its symmetry throughout the cine run (*B* and *D*). Single film could by symmetric or asymmetric depending on time taken.

FLATTENING

No fluid waves were present in any of the 5 patients with flattening. Flattening was associated with asymmetry of the opaque band (Fig. 2, *A* and *B*) in 2 patients proven to have constrictive pericarditis at surgery. Flattening was associated with symmetric thickened bands in 3 patients; it was never proven whether these patients had pericardial fluid, thickening or both.

ASYMMETRY

Asymmetry was present in 14 cases with and 4 cases without fluid waves (Fig. 3, *A* and *B*; 4, *A* and *B*; and 5, *A*–*D*). Two patients with constrictive pericarditis proven

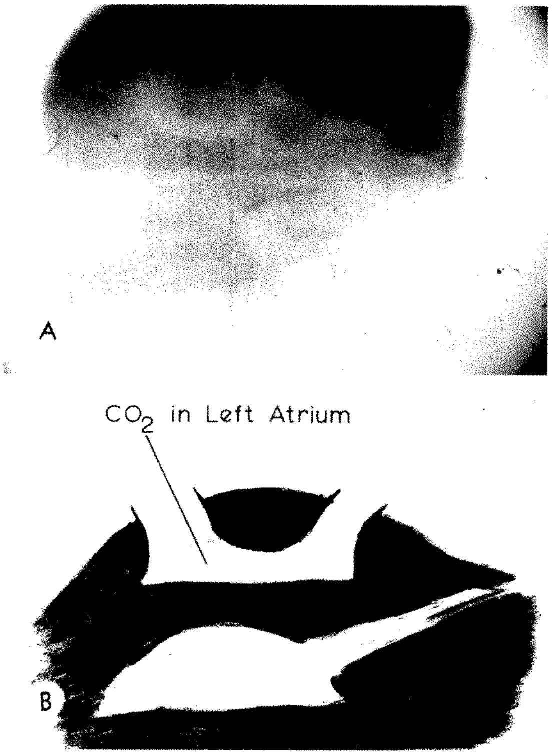

Fig. 6. (*A* and *B*) Pericardial effusion with carbon dioxide visualized in left atrium and pulmonary veins in patient with atrial septal defect.

at surgery had flattening, asymmetry and absence of a fluid wave. In the absence of flattening, asymmetry was of no specific diagnostic value.

GAS IN OTHER STRUCTURES

The superior vena cava was observed in 41 examinations. In 2 cases, the space between the superior vena caval gas shadow and the lung was increased in width. Both patients had right pleural effusions, 1 due to congestive failure and the other due to constrictive pericarditis.

In 25 examinations, gas was identified in the right ventricle and in 7 examinations it was seen in the right or main pulmonary artery. The inferior vena cava was visualized in 11 examinations and the hepatic veins in 4. The right atrial appendage was identified in 3 examinations. The left atrium and pulmonary veins were seen in a patient with an atrial septal defect (Fig. 6, *A* and *B*). The crista terminalis[9] was identified in 19 examinations.

SPURIOUS SHADOWS

Gas in the main stem bronchus, carina, and trachea occasionally was observed as a spurious gas shadow, but on cineroentgenography it was readily discerned from gas in the atrium, superior vena cava, right ventricle and pulmonary artery.

COMMENT

The advantages of recording carbon dioxide injections by means of cineroentgenography are many when compared to single film techniques. First, there are mechanical and technical advantages. The examination is done under fluoroscopic control. The filming is started before gas is

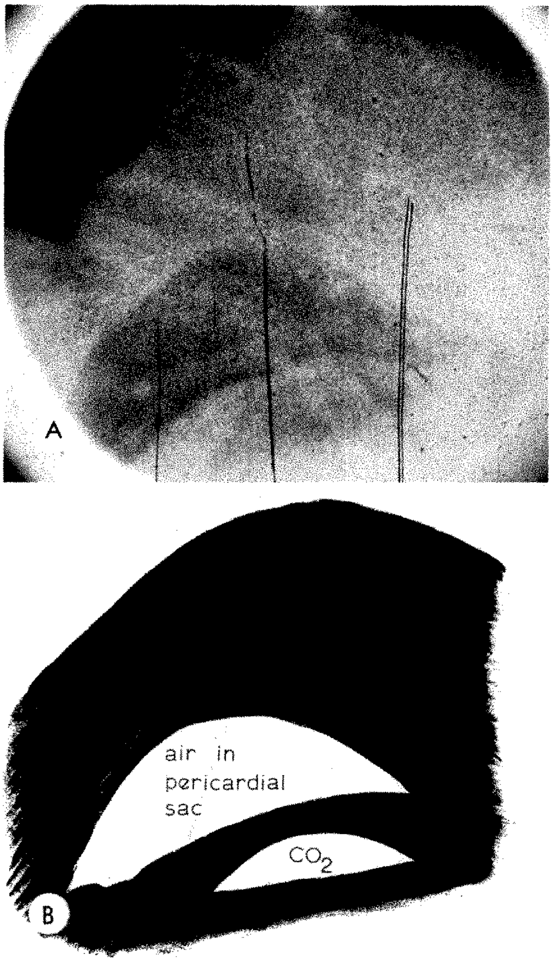

Fig. 7. (*A* and *B*) Absent fluid wave. Note air in pericardial sac and marked thickening of pericardium due to lymphomatous infiltrate. CO$_2$ in right atrium also reveals fluid present.

injected. Timing problems are automatically eliminated and a recording of gas in the right atrium is obtained in every patient regardless of the speed of arrival in or disappearance from the atrium. A satisfactory study has been obtained in a patient with superior vena caval occlusion by extending the examination to allow the gas to reach the atrium via collateral circulation to the inferior vena cava. Minor changes in the position of the patient can be made during the examination to assure optimal projection of the gas shadow and the associated anatomic structures of interest to the examiner. The examiner gets an immediate impression of the pathologic changes present and knows instantly whether more studies or a repeat injection are necessary. Small amounts of gas may be used. An injection of 50 cc. is almost always sufficient since timing is not a critical factor with this technique. With this small amount of gas, there is less reluctance to make a repeat injection when this is necessary or desirable. Finally, the decision about when to permit the patient to lie flat is easily made since the disappearance of the gas from the right atrium is readily seen fluoroscopically.

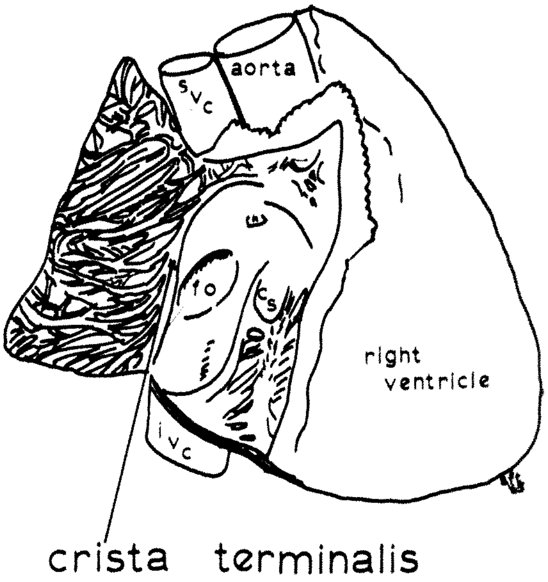

Fig. 8. Diagram of crista terminalis of the right atrium. SVC = superior vena cava; IVC = inferior vena cava; fo = foramen ovale; CS = coronary sinus.

Besides the technical advantages of the cine-procedure, there are inherent advantages in the interpretation of the studies when compared to single film techniques. These result from a continuous recording of events as they occur, providing many individual roentgenograms, plus the facility for observing the dynamic phenomena not only of the gas bubble itself, but also of the associated anatomic structures. Thus extraneous lung markings, bronchi, and pulmonary vessels are readily discerned from the cardiac border by the inter-relationship of their dynamic changes. The most important conclusion to be drawn after the demonstration of an opaque band more than 5 mm. thick is whether there is pericardial effusion or pericardial thickening. The visualization of a fluid wave indicates with a high degree of reliability that pericardial effusion is present. The absence of a fluid wave is indicative that there is pericardial thickening with or without the presence of small amounts of fluid (Fig. 7, *A* and *B*). Asymmetry of the thickened opaque band is not of any specific diagnostic significance except when it is associated with flattening of the band. The two together are strongly suggestive of constrictive pericarditis. Pleural effusion may give a false impression of pericardial effusion by causing an apparent thickening of the opaque band. Recording of the passage of carbon dioxide through the superior vena cava permits the diagnosis of pleural rather than pericardial effusion by observing the widening of the space between the cava and the lung. This is easily included in the cine-examination. With pleural fluid, there may also be thickening of the band about the pericardium during expiration and thinning on inspiration. These findings are readily observed on the cine-film.

The crista terminalis is a smooth muscular ridge which is situated mainly on the lateral wall of the right atrium. It begins on the upper part of the septum and after passing anterior to the orifice of the superior vena cava skirts the right margin of the orifice and then extends to the right side of

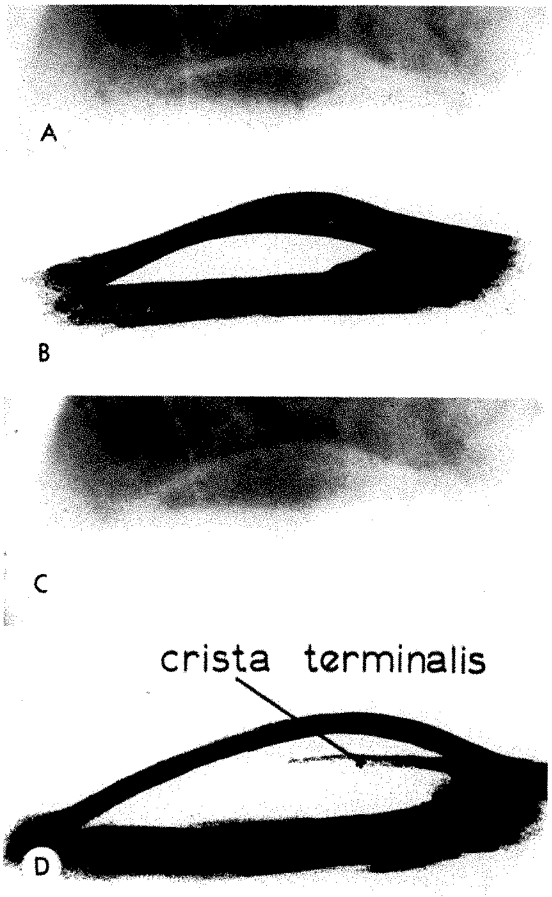

Fig. 9. (*A* and *B*) Apparent thickening of right atrial band due to all the gas in right atrium being trapped behind crista terminalis. (*C* and *D*) Same patient few seconds later with gas now in atrium proper demonstrating a normal atrial band.

the orifice of the inferior vena cava where it is connected to the right end of the valve of the latter vessel. It occupies the site of the embryologic right venous valve. It indicates the junction between the part of the heart derived from absorption of the right horn of the sinus venosus (sinus venarum—smooth walled) and the parts arising from the original atrium (trabeculated—rough walled).[3]

Cineangiography offers a frequent opportunity to visualize the crista terminalis (Fig. 8) of the right atrium, but, more important it clearly depicts the compartmentalization which can occur and avoids the false positive interpretation (Fig. 9, *A–D*) which is possible when, in a single film, all the gas in the right atrium is trapped posterior to the crista.

The safety of carbon dioxide angiography is well established. Carbon dioxide has certain advantages over other injection media: it is removed rapidly from the body so that injected amounts are not cumulative. It is a naturally present substance to which no sensitivity occurs. It causes no irritation to the veins and, therefore, causes no subsequent venous occlusion. Patients experience no unpleasant sensations with it and are, therefore, very cooperative during its injection.

We believe that the only contraindication to the performance of carbon dioxide angiocardiography is severe pulmonary disease in which carbon dioxide retention can cause difficulty to the patient. We do not believe that the presence of a left to right shunt contraindicates the procedure.[1,12] In the 1 patient in the series with an atrial septal defect, there was visualization of the left atrium and pulmonary veins, but the left lateral decubitus position caused the gas to remain trapped in the left atrium and pulmonary veins, permitting uninterrupted flow of blood through the mitral valve into the left ventricle.

We encountered no patients with pulmonary infiltrates adjacent to the heart. To our knowledge there were no false positive examinations due to the presence of pericardial fat.[15] We encountered no intra-atrial tumors in the 100 examinations. There were no patients in whom the left atrium protruded beyond the right atrial border.

SUMMARY

Carbon dioxide angiocardiography is a safe and simple diagnostic procedure to perform. It is more readily and more easily performed with cineroentgenography than with single film technique. One hundred studies were carried out in 90 patients. Cardiac pulsations were of no value in assessing the presence of pericardial fluid. The presence of a fluid wave in a thickened opaque band correlated well with the pres-

ence of pericardial effusion. The absence of a fluid wave in a thickened opaque band was indicative of pericardial thickening with or without the presence of small amounts of fluid. Asymmetry of the opaque band was of no specific diagnostic significance except when associated with flattening; this combination is strongly suggestive of constrictive pericarditis.

Compartmentalized gas due to the crista terminalis of the right atrium is clearly depicted and false positive interpretation which is possible in a single film, when all the gas in the right atrium is trapped posterior to the crista, can be avoided. Visualization of the superior vena cava is of value in a more accurate assessment of right pleural effusion.

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ANOMALOUS PULMONARY VENOUS RETURN, PULMONARY SEQUESTRATION, BRONCHIAL ATRESIA, APLASTIC RIGHT UPPER LOBE, PERICARDIAL DEFECT AND INTRATHORACIC KIDNEY

AN UNUSUAL COMPLEX OF CONGENITAL ANOMALIES IN ONE PATIENT

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THERE have been many congenital cardiovascular and respiratory defects described in the medical literature over the years. Some of these have been multiple anomalies. We were recently confronted with a unique combination of intrathoracic malformations which we believe merit reporting.

REPORT OF A CASE

M.T., a 44 year old white woman, was admitted to Ohio State University Hospital complaining of malaise, mild dyspnea and cough for the 6 months previous to admission. The patient denied night sweats, chills, weight loss, hemoptysis or chest pain. She reported frequent mild respiratory infections since childhood. In 1938 dextrocardia was diagnosed by physical examination and chest roentgenography. Previous to admission at Ohio State University, the patient had been seen at another hospital where a chest roentgenogram showed the dextrocardia, a questionable mass in the right hemithorax and possible collapse of the right lower lobe. At that time, bronchoscopy with biopsy was negative.

Physical examination at Ohio State University revealed a well developed, well nourished lady in no distress. A Grade III systolic murmur was heard over both sternal borders. A distinctly different Grade II systolic murmur was present in the same area. There were decreased breath sounds over the right lower lung field, accompanied by decreased excursion. The liver was felt slightly below the costal margin on the right.

Laboratory studies were within normal limits on admission. Chest roentgenograms were obtained. Dextroposition of the heart and a paravertebral lobulated mass above the right diaphragm were seen on the study (Fig. 1, *A* and *B*). There was also a generalized haziness over the lower half of the right lung field as seen on the posteroanterior roentgenogram. Air was noted under the diaphragm after a diagnostic pneumoperitoneum. The possibilities of sequestrated lung or tumor were entertained. Pulmonary function was somewhat depressed with a maximum breathing capacity of 46 per cent of predicted and a vital capacity of 81 per cent of predicted.

Because of the possible sequestration in the right hemithorax, retrograde thoracic aortography was carried out. The findings were interesting. The right kidney was seen herniated into the thorax apparently through the foramen of Bochdalek. The right renal artery arose in the normal position, and coursed superiorly to supply the thoracic kidney (Fig. 2). Adjacent and medial to the right kidney was a smaller mass which apparently had its blood supply from the abdominal aorta. The vessel originated at the renal artery level and went superiorly and posteriorly to supply this sequestrated right lower lobe (Fig. 2). A vein was seen descending vertically to the region of the middle third of the right diaphragm. This was assumed to be an anomalous venous return emptying into the inferior vena cava.

Subsequently, right heart catheterization with angiography was done (Fig. 3 and 4). Dextroposition and dextrorotation of the heart were well visualized. The left pulmonary arterial branches had normal distribution. On the right, the overexpanded middle lobe received branches from the pulmonary artery.

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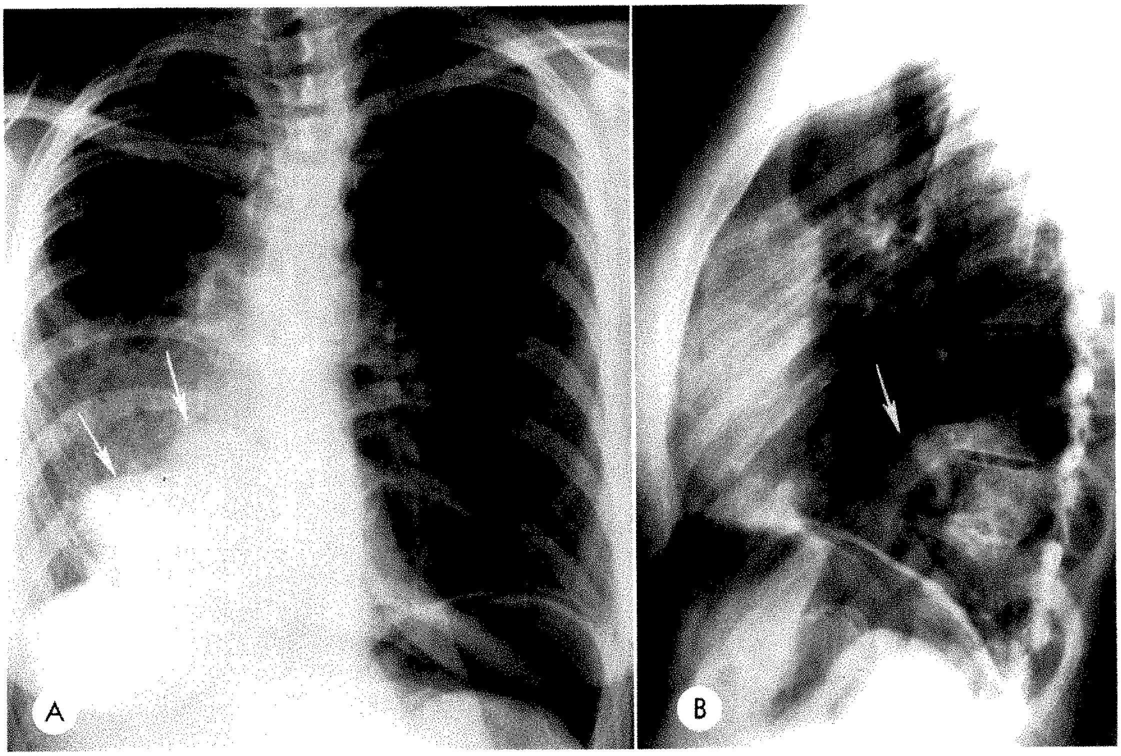

FIG. 1. (*A*) Anteroposterior and (*B*) lateral chest roentgenograms showing dextroposition and anterior displacement of the heart, and a lobulated paravertebral mass above the right diaphragm (arrows). The free air under the left diaphragm represents diagnostic pneumoperitoneum.

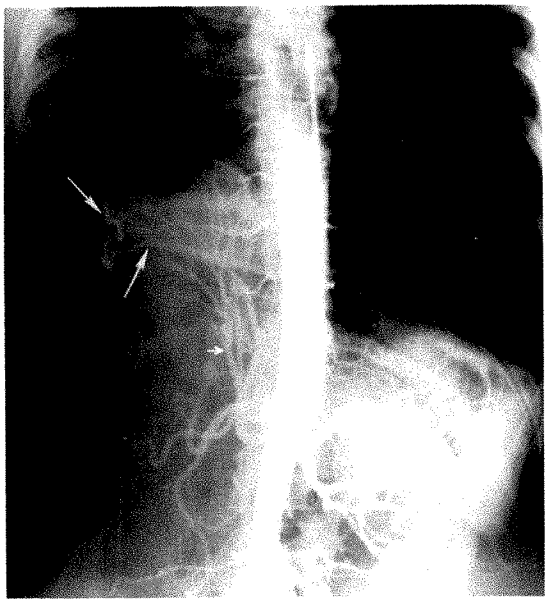

FIG. 2. Abdominal aortogram proves that part of the lobulated mass is a thoracic kidney supplied by the renal artery (short arrow). The rest of the mass and part of the right lower lobe received arterial blood supply through a branch of the abdominal aorta (long arrows).

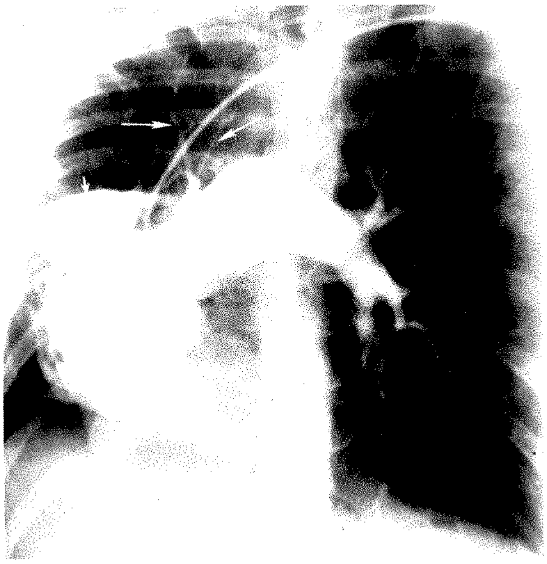

FIG. 3. The arterial phase of right heart angiography demonstrates dextroposition and rotation of the heart, and absence of the right upper and lower lobe pulmonary arteries. Only the branches to the overexpanded middle lobe are present (long white arrows). The right atrial appendage (short white arrow) is seen projecting free through an apparent defect in the pericardium.

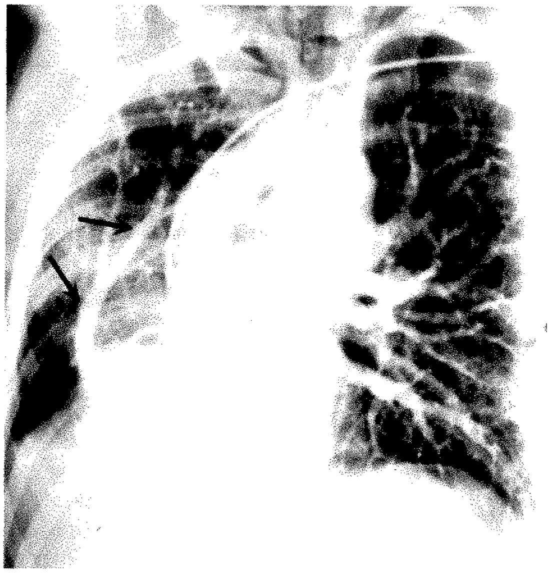

FIG. 4. Right heart angiogram, venous phase, illustrating a single anomalous pulmonary vein on the right, apparently returning to the inferior vena cava (arrows). The left atrium is small and the venous tributaries on the left are abundant.

However, the branches to the right upper and lower lobes were absent. In addition, part of the right side of the pericardium was apparently absent, allowing the atrium to project out, free of restraint. In the venous phase of the angiography, a large anomalous right pulmonary vein was seen passing from the apical region to the diaphragm and apparently into the inferior vena cava. Bronchography was done and showed atresia of the right upper lobe bronchus and aplasia of the right upper lobe in addition to the hypoplasia of the rest of the right bronchial tree (Fig. 5).

It was now obvious that this patient had sequestration of the right lower lobe with its anomalous arterial supply, a partial anomalous pulmonary venous return, atresia of the right upper lobe bronchus with aplasia of the right upper lobe, partially absent right side of the pericardium, intrathoracic kidney, and dextroposition and dextrorotation of the heart, probably secondary to the pulmonary pathology.

Since the patient had minimal symptoms, no surgical intervention was planned. She was reassured and discharged.

DISCUSSION

This case represents an uncommon collection of intrathoracic, cardiovascular and respiratory anomalies in a single patient. To our knowledge, they have not been described before as all occurring in one patient.

Individually, or in combinations these anomalies are interesting and important, not only from the present day medical, surgical and radiologic standpoints, but also historically.

In 1939, Winslow (quoted by H. Brody[1]) described drainage of pulmonary veins into the right side of the heart. Since then the anomalous pulmonary venous return has been reported quite often. Brody,[1] in 1942, reviewed all of the 100 cases in the literature to that time and presented 2 more of his own. We know now that an anomalous pulmonary vein can empty at a number of areas and is frequently accompanied by other vascular anomalies.[8] There may be accompanying dextroposition of the heart and the right lung may appear hypoplastic.[6] In 1956, Edwards and Helmholz[5] gave a classification of anomalous venous return depending on site of drainage and accompanying cardiac malformation. The two basic types of pulmonary venous drainage, complete and incomplete, are totally dif-

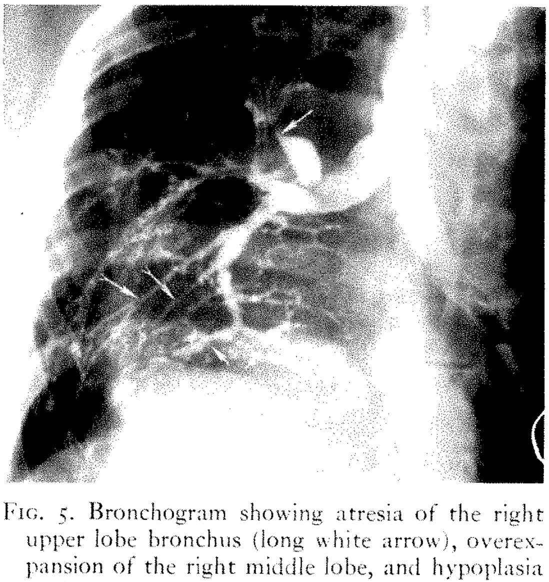

FIG. 5. Bronchogram showing atresia of the right upper lobe bronchus (long white arrow), overexpansion of the right middle lobe, and hypoplasia of the right lower lobe bronchi (feathered arrows). There is also separation of the basal segment bronchi by the described lobulated mass (short white arrow).

ferent in prognosis. Complete pulmonary venous drainage, without additional defect, is incompatible with life, returning no pulmonary blood to the left heart. Incomplete drainage is compatible with long life, particularly if the drainage empties into the inferior vena cava,[5] as in the case of our patient. Most cases with the vein emptying into the inferior vena cava are asymptomatic. However, there is a left to right shunt which on occasion is severe enough to cause considerable fatigue and dyspnea.[9]

Sequestration of the lung with its anomalous pulmonary artery was probably first noticed by accident. In 1940, Harris and Lewis[12] reported a fatal complication in a 5 year old girl undergoing a right lobectomy for pulmonary abscess. The lobe was sequestrated with an anomalous pulmonary artery from the aorta unrecognized. Subsequently, in 1946, Pryce[16] reported 7 cases of anomalous pulmonary artery with intralobar sequestration.

Sequestration with anomalous pulmonary arteries has been reported by many writers since.[2,3,10,14,15,18] Although occurring almost exclusively in the basilar segments of the lower lobes, it has been described rarely in the upper lobes.[17] It has been pointed out for some time that an anomalous pulmonary artery may supply (a) normal lung, (b) sequestered lung, and (c) both normal and sequestered lung.[4] The artery arises from the lower thoracic or upper abdominal aorta. In our patient it arose from the abdominal aorta and supplied both the sequestered lobe and a small portion of apparently normal lung. Angiography has become important in the diagnosis of these entities and is important as a preoperative work-up, as pointed out by Kenney and Eyler.[14] Cough, purulent sputum, fever and even severe bronchopneumonia may occur. If the complications are sufficiently severe and chronic, surgical excision of involved segments is the proper treatment.

Absence of a portion of the pericardium has been reported on numerous occasions. It accompanies diaphragmatic hernia in some cases and is unusually asymptomatic.[7,11,18]

SUMMARY

An unusual case is reported in which 6 distinct anomalies with 2 apparently secondary abnormalities were present. These have been demonstrated by means of angiography and bronchography.

By using bronchography and particularly angiography early, we were able to identify the thoracic abnormalities and avoid exploratory operation.

In addition, the roentgenologic appearance of the sequestration and of the anomalous pulmonary venous return, provided more than one possible explanation for the mild fatigue and minimal dyspnea the patient had.

Operative correction would entail a very radical procedure in view of the numerous anomalies present; however, since the patient's symptoms were minimal, it was not advised.

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CONGENITAL CORONARY ARTERIAL FISTULA TO A PERSISTENT LEFT SUPERIOR VENA CAVA*

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IN CORONARY arterial fistula, the coronary artery abnormally communicates directly with the different cardiac chambers, coronary sinus or pulmonary artery.[1,3] The present case is unique in that the arteriovenous fistula involves the posterior circumflex branch of the left coronary artery and a left superior vena cava.

REPORT OF A CASE

J.B. (G-NHC-H 44-11-67). This 43 year old, gravida 4, para 2, white female was admitted for evaluation of a cardiac murmur. A continuous murmur was incidentally discovered during her second pregnancy, 9 years previously. She had mild easy fatigability throughout her life which did not bother her significantly, so that she never sought medical attention.

Six years ago, her easy fatigability increased and was associated with intermittent fever for a period of approximately 4 months. She was confined for a period of 5½ weeks in another hospital for subacute bacterial endocarditis and cardiac failure. She was treated intensively with digitalis, mercurials and antibiotics.

Five and a half years ago, she underwent a posterolateral thoracotomy with a presumptive diagnosis of persistent ductus arteriosus. Instead, a ligamentum arteriosum was found and ligated. A left superior vena cava was seen where a thrill was felt. Proximal occlusion of the left superior vena cava did not change the character of the thrill. A continuous murmur was audible over the left superior vena cava and pulmonary artery. A brief exploration failed to elucidate the cause of the thrill and murmur. Without opening the pericardium, the operation was terminated.

Five years ago, she had had intravenous angiocardiography and right heart catheterization. The significant findings in the catheterization data (Table I) were a step-up in oxygen content of the right atrial sample from that of the right superior vena cava. Three years ago, another right heart catheterization failed to delineate the lesion. One year ago, she was readmitted because of increasing easy fatigability and palpitations. Further studies were done which included retrograde aortography and left ventriculography.

At the age of 5, she had had empyema from pneumonia of the left lower lung. Tonsillectomy and an adenoidectomy had been performed at the age of 10 years.

Physical Examination. This revealed a well developed, well nourished female in no distress with a pulse of 90/min. and blood pressure of 126/80 mm. Hg in the right arm. Pertinent findings were centered in the heart, which was slightly enlarged to percussion, although the point of maximal impulse was at the 5th interspace in the left mid-clavicular line. The second sound was louder at the aortic area than at the pulmonic area. A Grade II–III/IV continuous machinery-like murmur with systolic and diastolic accentuations was maximally heard at the 3rd interspace of the left parasternal region. The rest of the examination was normal.

Laboratory Examination. Blood studies, urinalysis and circulation time were normal except for 17.9 units bromsulfonphthalein retention in 45 minutes. The electrocardiogram showed minor nonspecific ST-T segment changes with no ventricular hypertrophy.

Roentgenographic Examination. Chest roentgenograms with barium swallow (Fig. 1, *A* and *B*) showed minimal cardiomegaly without specific chamber enlargement. The ascending aorta was moderately prominent. The pulmonary vascularity was normal and the lungs were clear.

Intravenous angiocardiograms (Fig. 2, *A* and *B*), from the left antecubital vein, showed bilateral superior venae cavae with a communicating innominate vein. The superior half of the left superior vena cava was narrow and half the size of the inferior portion. There was a mixing defect in the left vena cava, about 4 cm.

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Table I

CATHETERIZATION DATA

Catheter Position		Oxygen Content (volume per cent)	Oxygen Saturation (per cent)	Pressure (mm. Hg)
Right superior vena cava		14.5	74.1	
Right atrium	—mid	14.3	72.8	5/2
	—outflow	16.7	85.2	
Right ventricle	—inflow	15.5	79.1	22/1
	—outflow	15.5	79.0	
Pulmonary artery		15.8	80.5	20/7
Femoral artery	—room air	19.0	96.3	126/70
	—breathing 100% oxygen for 3 minutes	20.8	101.4	

above its junction with the coronary sinus. The dextro- and levo-angiocardiographic phases were normal except for a dilated ascending aorta. The coronary arteries were poorly visualized.

Left ventriculography and retrograde aortograms (Fig. 3, *A* and *B*) demonstrated neither a ventricular septal defect nor aortic valvular insufficiency. The ascending aorta was dilated. The right coronary artery was normal. The anterior descending branch of the left coronary artery was not identified. The posterior circumflex branch was dilated and very tortuous. Its distal ramifications were not visualized. There was immediate faint opacification of the left superior vena cava with the flow directed through the coronary sinus to the right atrium.

Operative Findings. Immediately visible on opening the pericardium was a dilated left coronary artery which coursed posteriorly and inferiorly to the tip of the left atrial appendage. After sharp dissection in this region, it was apparent that a branch of the posterior circumflex artery, 8–9 mm. in diameter, communicated with a persistent left superior vena cava. Branham's sign was positive. The fistula was then divided and both ends were repaired.

Follow-up. The continuous murmur disappeared after surgery. She has been asymptomatic for the last 2 years. Chest roentgenograms have shown no change in the prominent ascending aorta and minimal cardiomegaly.

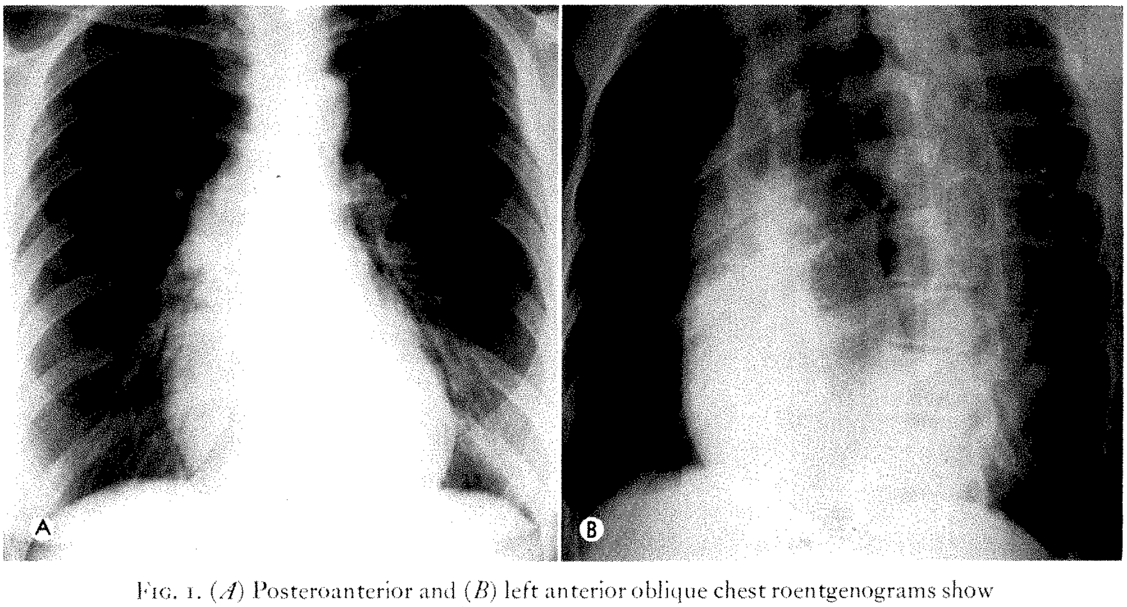

Fig. 1. (*A*) Posteroanterior and (*B*) left anterior oblique chest roentgenograms show minimal cardiomegaly and a very prominent ascending aorta.

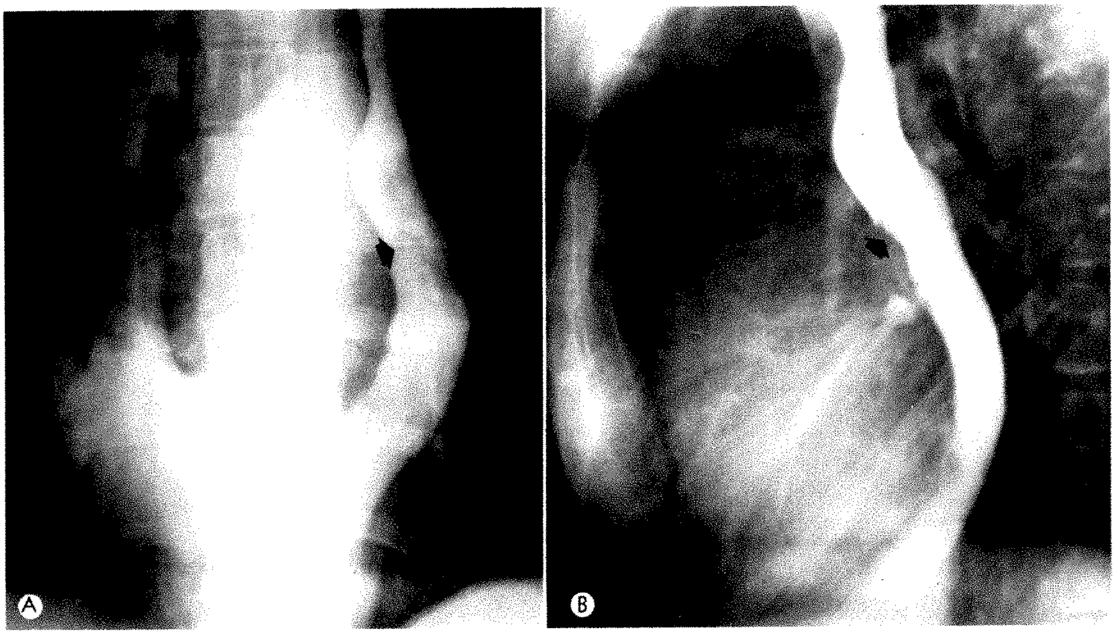

Fig. 2. (*A* and *B*) Intravenous angiocardiograms demonstrate bilateral superior venae cavae. An indirect sign of the coronary arterial fistula is seen as a mixing defect (arrows) in the central and anterior aspect of the left superior vena cava.

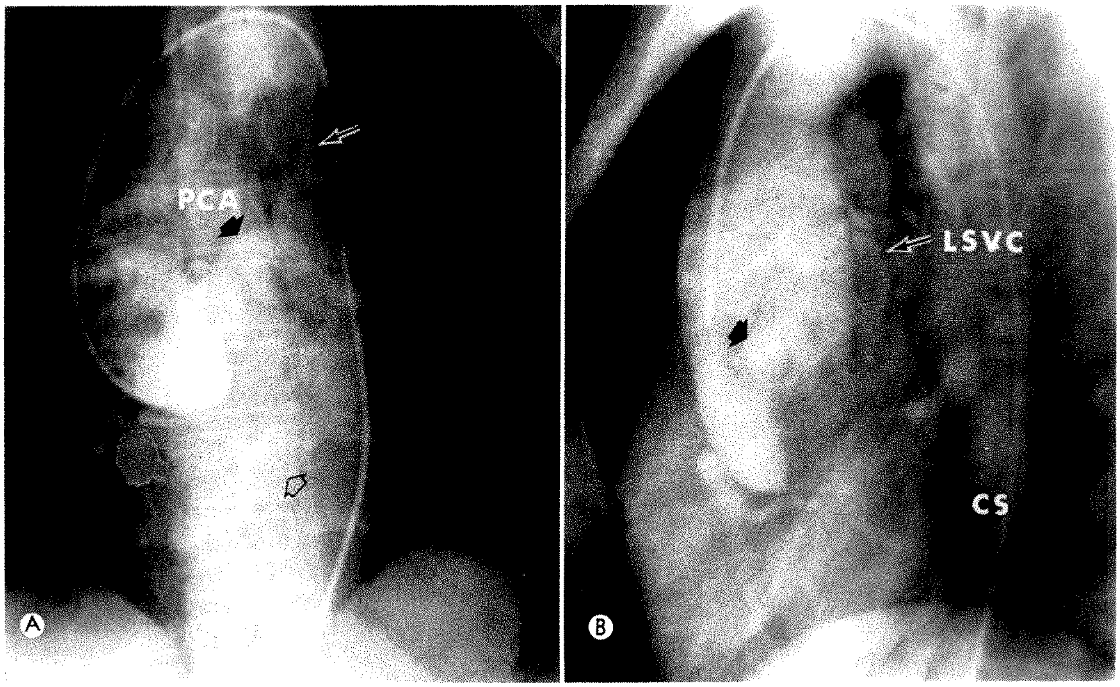

Fig. 3. (*A* and *B*) Biplane retrograde aortograms reveal a tortuous and dilated posterior circumflex coronary artery (PCA) as compared to the normal right coronary artery. A left-to-right shunt is shown by immediate faint opacification of the left superior vena cava (LSVC) and coronary sinus (CS).

DISCUSSION

A precordial continuous murmur is a consistent sign of coronary arterial fistula. The location and intensity of the murmur depend on the site and degree of the shunt. Signs of left-to-right shunt may be manifested as right heart enlargement, prominent pulmonary artery segment and increased pulmonary vascularity in the chest roentgenograms. In this and other cases,[1] a dilated ascending aorta was present.

The evidence for left-to-right shunt is present in the arteriovenous fistula where the coronary artery communicates with the coronary sinus, right atrium, right ventricle, or pulmonary artery. Since the coronary sinus and left superior vena cava were not catheterized in this case, only the approximate site and degree of the shunt are known. Although cardiac catheterization does not indicate the anatomic cause of the left-to-right shunt, its precise location and magnitude can be determined in most instances.

In the rarer type of arteriosystemic fistula where the coronary artery communicates with the left atrium or left ventricle, the hemodynamic pattern of aortic insufficiency occurs without evidence of a left-to-right shunt. Right cardiac catheterization in these cases is noncontributory. Chest roentgenograms may reveal left ventricular enlargement and aortic dilatation.

The lesions frequently considered in the differential diagnosis are: persistent ductus arteriosus, aorticopulmonary window, aortic sinus of Valsalva aneurysm, aortic valvular regurgitation, ventricular septal defect with aortic insufficiency, anomalous origin of the coronary artery from the pulmonary artery, atrial septal defect with mitral stenosis, and partial anomalous pulmonary venous return. The pathophysiology has been extensively discussed by Gasul and associates.[1]

The definitive examination is selective coronary arteriography or retrograde aortography. It delineates the involved coronary artery and the site of the shunt to best advantage. In the case presented, aortograms demonstrated the fistula from the posterior circumflex branch of the left coronary artery to the persistent left superior vena cava. In retrospect, the lesion is shown as a mixing defect in the left superior vena cava by intravenous angiocardiography.

The majority of patients with coronary arterial fistula have been asymptomatic. However, congestive failure, thrombosis and embolism, as well as bacterial endocarditis may occur as complications.[3] The lesion is correctable and must be treated surgically.[2]

SUMMARY

A case of congenital coronary arteriovenous fistula of the left coronary artery (posterior circumflex branch) to a persistent left superior vena cava is reported. The lesion was diagnosed preoperatively by retrograde aortography. Successful surgery led to complete recovery.

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ROENTGENOGRAPHIC ANATOMY OF HUMAN CORONARY ARTERIES*

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ACCURATE identification of coronary arterial branches is important in the interpretation and description of coronary arteriograms, especially if surgery is being considered. Learning the roentgenographic anatomy was found to be difficult. Despite careful and complete descriptions in the anatomic literature, descriptions and photographs of the arteries in the exact projections used in roentgenography are rare, and may not include variations in different phases of the cardiac cycle.

The authoritative and beautifully illustrated book by James[1] has been most helpful. However, gaining a thorough understanding of the anatomy required comparison of photographs in James' book with 3 dimensional vinylite corrosion casts of human coronary arteries. These casts were turned to the proper projection and were compared with coronary arteriograms.

Conclusions drawn from the study of 55 of these corrosion casts, information obtained from Drs. Sones and Shirey, and coronary arteriographies personally performed on 250 patients are discussed herein.

TECHNIQUE

Selective cine coronary arteriography was performed with individual catheterization of the right and left coronary arteries, following the method of Sones and Shirey[8] as closely as possible. Contrast medium was injected in the left and right anterior oblique projections, and sometimes in the posteroanterior also. Roentgenography usually was done while the patient held a deep breath. The technique has recently been described in detail.[3]

Single frames shown herein were reproduced from the 35 mm. film with apology for the fact that single frames can never show as much as viewing the pictures in motion, and can actually be misleading diagnostically.

FINDINGS

RIGHT CORONARY ARTERY—LEFT ANTERIOR OBLIQUE PROJECTION (Fig. 1 through 4)

The right coronary artery follows the atrioventricular groove and is near the right border of the cardiac shadow during diastole. The midportion of the right coronary artery is usually somewhat inside the shadow during systole. The artery goes toward the patient's right initially. It turns downward until it reaches the acute margin. A second bend is usually apparent at the acute margin, and the artery usually passes beneath the shadow of the diaphragm for the remainder of its course, unless the heart is vertical and the patient can take a very deep breath. The right coronary artery usually follows the atrioventricular groove to the crux (the point where the atrioventricular, interventricular, and interatrial grooves meet on the posterior aspect of the heart). The right coronary artery usually curves (Fig. 1, *A* and *B;* and 2, *A* and *B*) upward just before reaching the crux, and often extends a short distance to the left of it in the atrioventricular groove (Fig. 3, *A* and *B*). It rarely goes as far as the obtuse margin. The distal right coronary artery occasionally gives collateral channels to the distal circumflex on the left of the crux.

Branches of the right coronary artery are rather variable. The conus branch is sometimes seen arising very near the origin and goes towards the left. It sometimes arises from the sinus of Valsalva as a sepa-

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* From Veterans Administration Hospital, Oteen, North Carolina.
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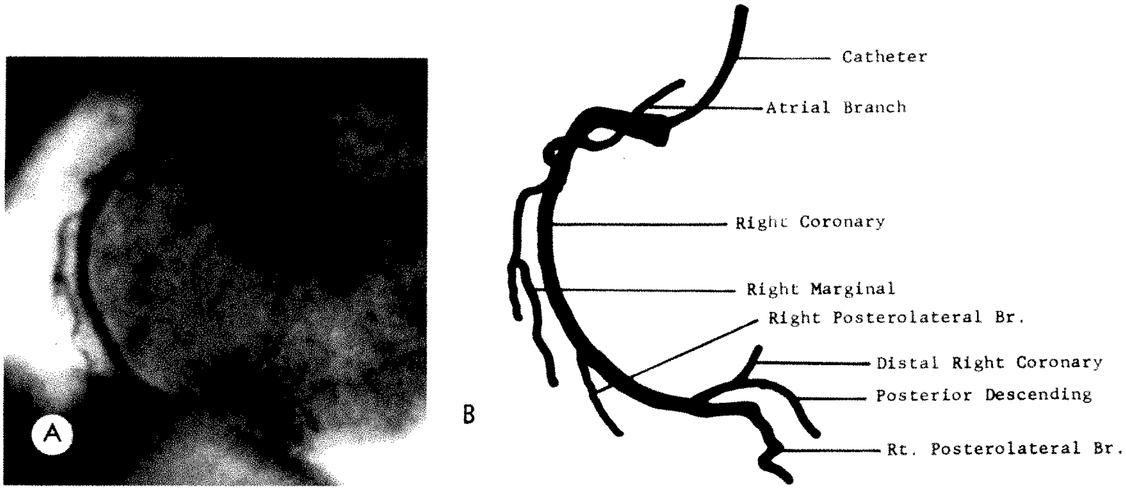

FIG. 1. (*A* and *B*) The right coronary artery in the left anterior oblique projection.

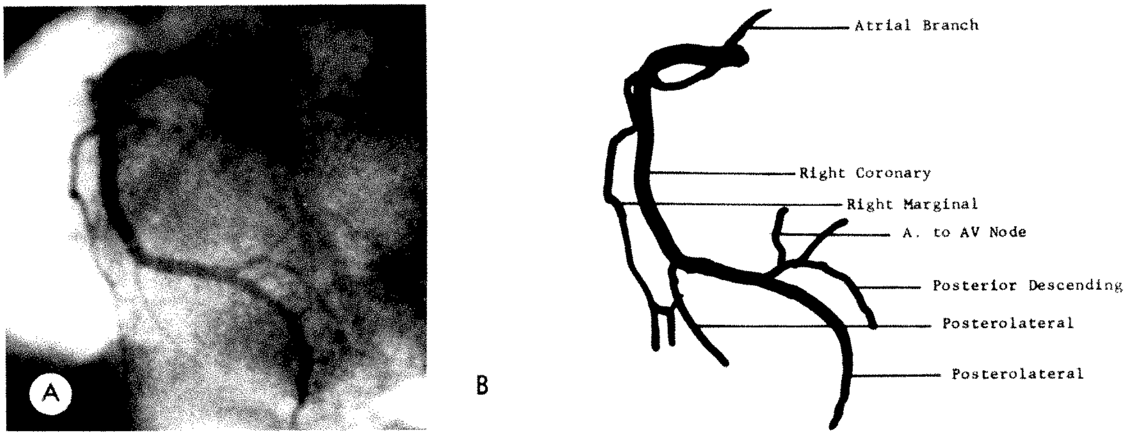

FIG. 2. (*A* and *B*) The same right coronary artery as in Figure 1, *A* and *B* showing the effects of a deep breath.

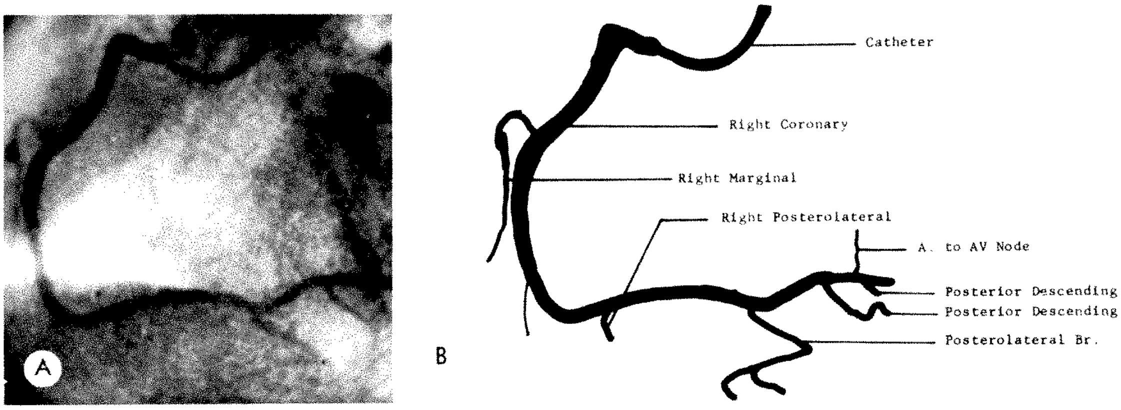

FIG. 3. (*A* and *B*) The right coronary artery of another patient in the left anterior oblique projection.

rate opening, and in that case would probably not be opacified by an injection directly in the origin of the right coronary. It may be a source of collateral channels to the anterior descending.

The artery to the sino-auricular node arises from the right coronary in about half of the cases. It arises from the first 1 cm. or 2 cm. and goes obliquely upward and to the left (Fig. 1, *A* and *B;* and 2, *A* and *B*). It is occasionally quite prominent and may rarely be a source of collateral channels to the circumflex through the left atrial branch.

One or two anterior right ventricular branches are usually found. They arch upward into the right for a short distance and then go downward to the left. They vary considerably in number, size, and position. These may also give collateral channels to the anterior descending artery.

There are usually 1 and sometimes 2 moderate sized or large right marginal arteries, which arise from the second or third quarter of the distance between the origin of the right coronary artery and the acute margin (Fig. 1 through 3). They curve irregularly and usually go downward near the right edge of the cardiac shadow. They often overlap the midportion of the right coronary artery during part of the cardiac cycle.

There is often a right posterolateral branch of the right coronary artery arising near the acute margin and passing to the left and slightly downward. It is often completely beneath the diaphragmatic shadow and can be seen only if the technique used will provide visualization through the diaphragm.

Occasionally, this posterolateral branch is equal to or larger than the distal right coronary artery. Such a branch usually crosses the posterior right ventricle and reaches the posterior interventricular groove (Fig. 2, *A* and *B*). It, therefore serves areas normally supplied by the posterior descending artery, which in this instance would probably be very small.

The artery to the atrioventricular node in the upper portion of the septum is usually seen as a very small artery going straight upward and identifies the crux (Fig. 3, *A* and *B*).

There is commonly a large posterior descending artery arising proximal to the crux and another arising distally (Fig. 3, *A* and *B*). There may be one in the posterior interventricular groove. There are sometimes as many as 4 medium size posterior descending arteries located to the right, left, and/or in the groove in any combination of sizes and locations. The posterior descending artery passes downward and perhaps slightly to the left at first. It usually is coming directly at the observer in the mid or distal portion. Here, it is likely to be beneath the diaphragmatic shadow, particularly if the patient cannot or does not take a very deep breath. A deep breath may improve visualization markedly, as in Figure 2*A*.

The distal posterior descending artery is occasionally connected to the distal anterior descending artery by a collateral channel going around the apex. The direction of movement of blood through this collateral channel would be toward the artery with the more severe obstruction.

The posterior portions of the right and left ventricle are supplied by the circumflex artery in about 10 per cent of patients. In these instances the right coronary artery ends between the acute margins and the crux without giving any large posterolateral branches (Fig. 4, *A* and *B*).

RIGHT CORONARY ARTERY—RIGHT ANTERIOR OBLIQUE PROJECTION (Fig. 5, *A* and *B*)

The proximal portion of the right coronary artery is seen largely on end in this projection. The midportion is well seen. It usually moves downward and slightly toward the spine. The portion distal to the acute margin is likely to also be seen more or less on end.

A conus branch, the anterior right ventricular branches, and the right marginal branches go anteriorly, and are more or less parallel to each other.

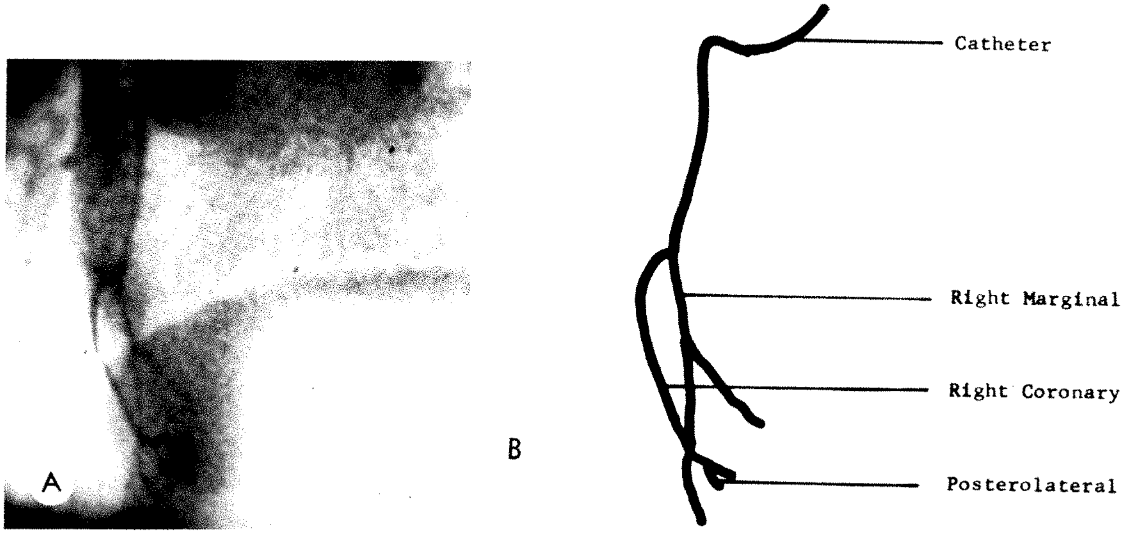

FIG. 4. (*A* and *B*) The right coronary artery of a patient with left coronary preponderance in the left anterior oblique projection.

If there is only one posterior descending artery present, the portion that is not below the diaphragm will probably be well seen. However, several posterior descending arteries are usually largely superimposed on each other. They may separate somewhat during some portion of the cardiac cycle. This view may demonstrate a constriction in the proximal posterior descending artery better than the left oblique projection, but such lesions are very rare.

The right anterior oblique projection of the right coronary artery, therefore, is unlikely to give much useful information which cannot be obtained in the left anterior oblique projection, and was usually omitted in the coronary arteriographies in this series.

LEFT MAIN CORONARY ARTERY—LEFT ANTERIOR OBLIQUE PROJECTION (Fig. 6, *A* and *B*; and 9, *A* and *B*)

This artery is usually large in diameter and its length is less than the diameter. It

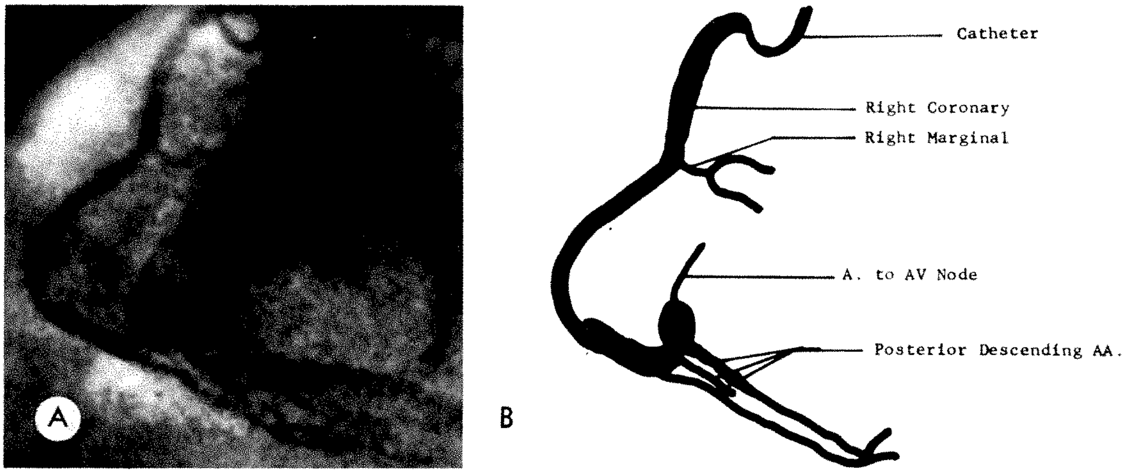

FIG. 5. (*A* and *B*) The same right coronary artery as in Figure 3, *A* and *B* seen in the right anterior oblique projection.

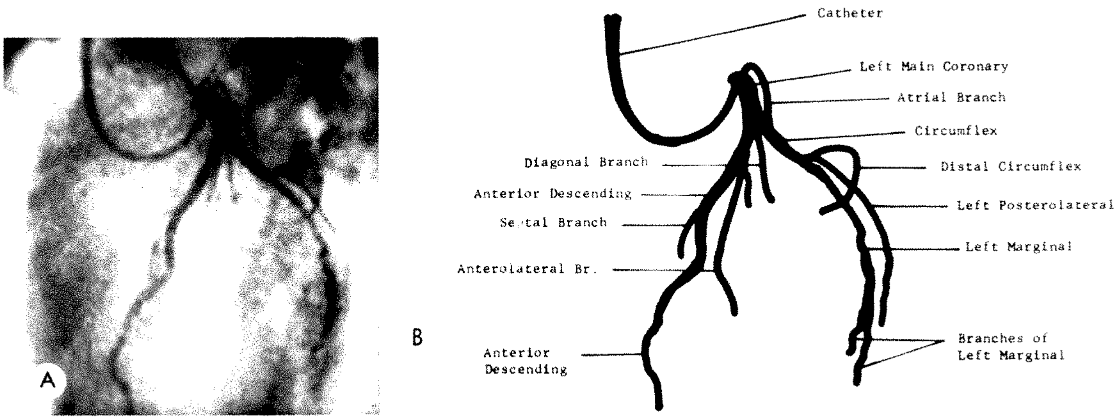

Fig. 6. (*A* and *B*) A left coronary artery in the left anterior oblique projection.

is generally seen well only in the left anterior oblique projection, and may be overlapped by the sinus of Valsalva or the anterior descending artery if the amount of obliquity varies slightly. The region of its branching to 2, 3, or more arteries is difficult to demonstrate well. Sometimes a slight change in the degree of obliquity helps considerably and injections are routinely done in at least 2 left oblique positions.

Occasionally, the left main coronary artery is nonexistent and the anterior descending and circumflex arteries arise from separate orifices in the sinus of Valsalva. They must then be catheterized separately for adequate demonstration arteriographically. Occasionally, because of layering and streaming of the medium, an injection appears to opacify all of the left sinus of Valsalva but will show only the circumflex or the anterior descending artery even though the other is present and normal.

ANTERIOR DESCENDING ARTERY—LEFT ANTERIOR OBLIQUE PROJECTION (Fig. 6, *A* and *B*; and 9, *A* and *B*)

The anterior descending artery usually passes downward at an angle of 20 to 45 degrees to the right. It tapers gradually as it approaches the apex. The proximal portion of the anterior descending artery is seen almost on end in this projection but the midportion is well seen. It can easily be followed to the apex or to the point where it disappears into the diaphragmatic shadow. The distal end is not usually seen well in this projection if it passes around the apex, as it commonly does. It may give a collateral channel to the posterior descending artery.

A single septal artery is usually seen in this projection. It appears as a long thin artery which tapers slowly; it goes $\frac{1}{4}$ to $\frac{1}{2}$ the distance to the apex. Occasionally, multiple septal arteries are seen in this view. The origins are usually obscure, because of overlapping of the anterior descending. The septal artery is almost parallel to the anterior descending but diverges slightly to the right as it progresses downward.

The diagonal branch approximately bisects the angle between the anterior descending and circumflex arteries. It may arise from the anterior descending artery, the circumflex artery, or may originate between the two. The exact site of origin is difficult to determine from this projection if it arises from the proximal 2 cm. of the anterior descending artery. It sometimes arises more distally along the anterior descending artery. If large, it usually bifurcates one or more times and approaches the apex. It rarely gives collateral channels to the left marginal branch of the circumflex artery.

There are several anterolateral branches arising from the anterior descending artery along its course. They pass downward,

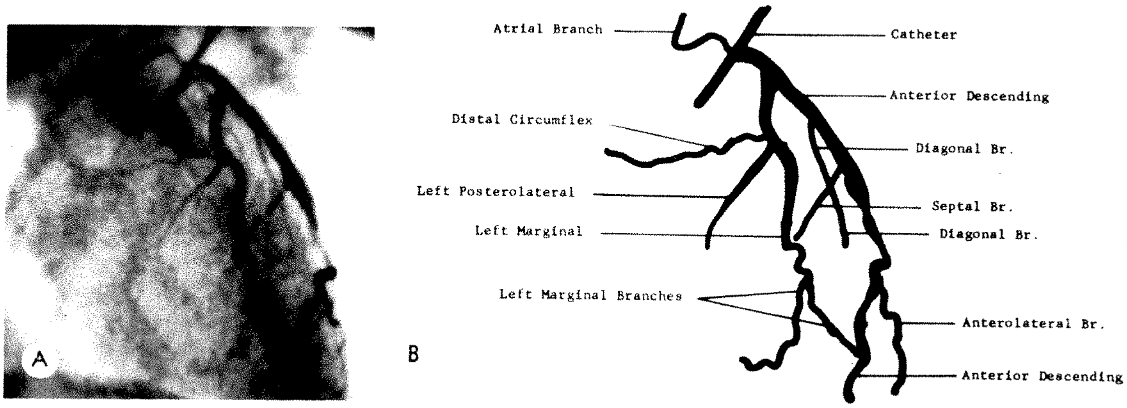

FIG. 7. (*A* and *B*) The left coronary artery of the same patient as in Figure 6, *A* and *B* seen in the right anterior oblique projection.

or downward and to the left. The number, size, and location of these branches vary greatly. There is normally either a series of several anterolateral branches of the anterior descending artery along its course, a very large single diagonal branch, or a left marginal artery in an unusually anterior position. The anterolateral branches from the midportion of the anterior descending artery usually do not overlap their parent artery much in this projection. Their origins generally are well demonstrated, which is important because most constrictions of these branches are in the first 1–2 mm.

ANTERIOR DESCENDING ARTERY—RIGHT ANTERIOR OBLIQUE PROJECTION (Fig. 7, *A* and *B*; and 10, *A* and *B*)

The proximal third of the anterior descending artery is seen well only in this projection. What appears to be a length of several millimeters in the left anterior oblique projection is seen as several centimeters in the right anterior oblique projection. Constrictions of the anterior descending artery are often located proximal to the septal artery or immediately distal to it, and therefore show well only in this projection.

The proximal third of the anterior descending artery is near the left cardiac border and it usually curves away from it a little as it progresses downward. It often crosses the left marginal artery, as shown in Figure 7, *A* and *B*. It can often be seen going to and around the apex.

One large and sometimes several medium sized septal arteries are usually seen in this projection. These arteries arise from the anterior descending artery at approximatey right angles.

Most of the diagonal and anterolateral branches from the anterior descending artery overlap the proximal half of their parent artery in most, but rarely all, of the cardiac cycle. The origins of these branches are likely to overlap it throughout the entire cycle. Anterolateral branches from the midportion of the anterior descending artery are often closer to the cardiac border than the parent artery in this projection, but can be farther from it.

The right anterior oblique projection, therefore, is much better for demonstration of the proximal half of the anterior descending artery itself but usually is not as helpful as the left anterior oblique projection for the diagonal and anterolateral branches.

CIRCUMFLEX ARTERY—LEFT ANTERIOR OBLIQUE PROJECTION (Fig. 6, *A* and *B*; and 9, *A* and *B*)

The proximal circumflex artery is seen only in the left anterior oblique projection and a good view of the first 1 to 3 mm. is often obtained only in a very narrow range of the degree of obliquity. Unfortunately, this is frequently a site of constrictive lesions.

The circumflex artery runs in or near the atrioventricular groove. It usually extends to the posterolateral region of the heart between the obtuse margin and the crux. It may terminate before reaching the obtuse margin. In a minority of cases, it goes to or beyond the crux and gives a posterior descending artery in addition to or instead of that arising from the right coronary artery (Fig. 9, *A* and *B*). It gives a collateral channel to the distal right coronary artery.

The origin of the posterior descending arteries from the right coronary artery has been commonly referred to as "right coronary preponderance" and a posterior descending artery coming from both the right coronary artery and the circumflex artery is called "balanced circulation." The contribution by the circumflex artery of most or all of the posterior descending arteries is called "left coronary preponderance." These terms are well established but must be used with the understanding that even in "right coronary preponderance," the vast majority of the arterial supply to the left ventricle still comes from branches of the left coronary artery, and left coronary disease is more important clinically.

Branches of the circumflex artery are quite variable. The artery to the sinoauricular node arises from the proximal few centimeters of the circumflex artery in about half of the cases. It passes upward and slightly to the right. Other atrial arteries, such as Kugel's artery, are occasionally seen passing to the left and somewhat upward. A left atrial circumflex artery occasionally arises near the origin of the circumflex artery and runs nearly parallel to it in or on the atrial side of the atrioventricular groove. It supplies the anterior aspect of the left atrium.

As stated above, a large diagonal branch may arise at or near the origin of the circumflex artery. It is likely to overlap the circumflex artery in all projections, and lesions at the origin of either are difficult to see. Fortunately, this is rather rare.

There is usually a small or moderate sized anterolateral branch from the first 2 or 3 cm. near the circumflex artery. It passes to the left and downward at perhaps a 45 degree angle.

A left marginal artery is nearly always present and is usually large. It sometimes is the terminal branch of the circumflex artery, although more commonly the circumflex artery continues for several centimeters beyond it as a small vessel. The left marginal artery may arise near the origin of the circumflex artery and distinction between an anteriorly displaced left marginal artery and an unusually lateral diagonal branch is arbitrary. The left marginal artery may arise as far laterally as the obtuse margin.

If the left marginal artery is large and is located far anteriorly, the diagonal branch is usually small and often arises some distance from the origin of the anterior descending artery. However, such hearts should normally also have a large left posterolateral branch from the distal circumflex artery or the distal right coronary artery.

The proximal or midportion of the left marginal artery is likely to overlap the circumflex artery in the region of the obtuse margin during much of the cardiac cycle and may be difficult to distinguish from it. It is helpful to remember that the distal circumflex artery usually moves slightly outward during the beginning of systole and the marginal artery moves inward during this period. The location of the atrioventricular groove is given by the opacification of the coronary sinus which follows a good injection of the left coronary artery and this helps in the identification of the distal circumflex artery.

The circumflex artery occasionally gives moderate sized or large branches anywhere between the obtuse margin and the crux. Those closer to the margin are called posterolateral branches and those near the crux are called posterior descending arteries. The dividing line between the two is somewhat arbitrary.

Distal anterolateral branches from the

circumflex artery, the left marginal and posterolateral branches are all near the left cardiac border in the left anterior oblique projection and can largely overlap each other during much of the cycle. Although they may appear to be quite close together, they are actually separated rather widely in space.

CIRCUMFLEX ARTERY—RIGHT ANTERIOR OBLIQUE PROJECTION (Fig. 7, *A* and *B*; and 10, *A* and *B*)

The proximal circumflex artery is seen on end in this projection. The portion of the circumflex artery around the obtuse margin is well seen. The circumflex artery passes approximately 45 degrees downward and to the right. Whether it is in or on the ventricular side of the atrioventricular groove can be easily determined when the coronary sinus opacification occurs. If the circumflex artery extends to the crux the distal portion is also seen on end.

Atrial branches, if present, make approximately right angles with the circumflex artery and pass upward and to the spine.

Proximal anterolateral branches from the circumflex artery overlap the diagonal branch and/or the proximal anterior descending artery. The left marginal artery and posterolateral branches may arise at a right angle from the circumflex artery, but they make an acute angle with the distal atrioventricular groove.

The left marginal artery usually appears quite large in this projection. Its average location is about halfway between the left cardiac border and the diaphragm and it heads approximately toward the apex. Its position varies considerably, and the right anterior oblique projection shows whether it is more anterolateral or posteromedial than usual.

Posterolateral branches are well seen in the right anterior oblique projection. They roughly parallel the left marginal artery proximally and tend to converge toward it distally.

The proximal portion of any posterior descending artery arising from the circumflex artery usually is seen well in this projection if the patient moves his diaphragm downward sufficiently. It is more difficult to see the distal portion.

ANTERIOR DESCENDING AND CIRCUMFLEX ARTERIES IN POSTEROANTERIOR PROJECTION (Fig. 8, *A* and *B*)

The posteroanterior projection is not satisfactory and should never be used as a substitute for the left anterior oblique or the right anterior oblique projections.

Early in the series, the posteroanterior projection was omitted entirely. More recently, it has been included in the routine coronary arteriographies because it is sometimes helpful for tracing the relationship between artery branches as the patient is turned from the left anterior oblique to the right anterior oblique position. Occasionally, it is also helpful if a lesion is suspected but not confirmed in the other views.

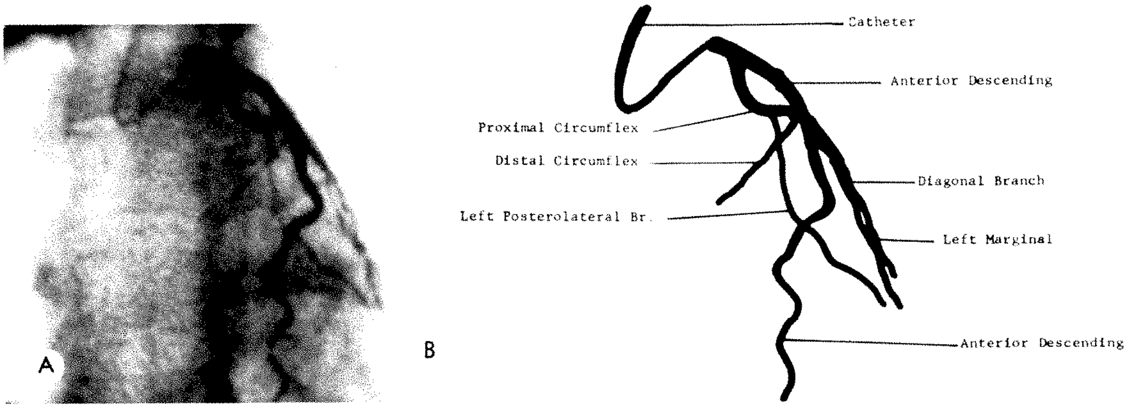

FIG. 8. (*A* and *B*) The same left coronary artery as in Figure 6, *A* and *B* and 7, *A* and *B* seen in the posteroanterior projection.

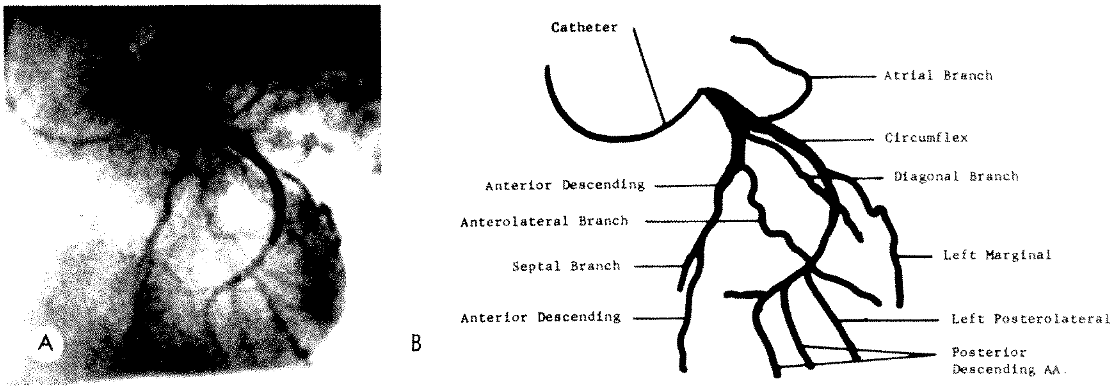

Fig. 9. (*A* and *B*) The left coronary artery of a patient with left coronary preponderance in the left anterior oblique projection.

Although most of the left coronary branches do not overlie the spine in the posteroanterior projection, radiation scatter from it is usually a problem and interferes with sharpness. The great variation in radiodensity in various portions of the field is also a problem.

The circumflex and anterior descending arteries, and their large branches usually more or less overlie one another in the posteroanterior projection. Identification of individual branches was usually possible, though tedious, by repeated comparison with other views. The proximal circumflex artery may often be identified by the fact that it moves considerably more than the anterior descending artery during systole.

DISCUSSION

The size and location of various branches of the coronary arterial system vary tremendously. A recent attempt to set up mathematical criteria for normal limits which could be coded on punch cards in a manner acceptable to statisticians was notably unsuccessful.

Occasionally, a branch becomes occluded at its origin without leaving a tell-tale stump and it is difficult to decide whether a branch was present and occluded by disease, or was never present. The solution of the problem appears to be largely a matter of judgment. It is helpful to remember that the sole function of the coronary arterial system is to transplant fluid, and cine arteriography shows the size of these conduits and the velocity of movement through them. If a large portion of the left ventricle is found to have no blood supply from any source in a technically good coronary arte-

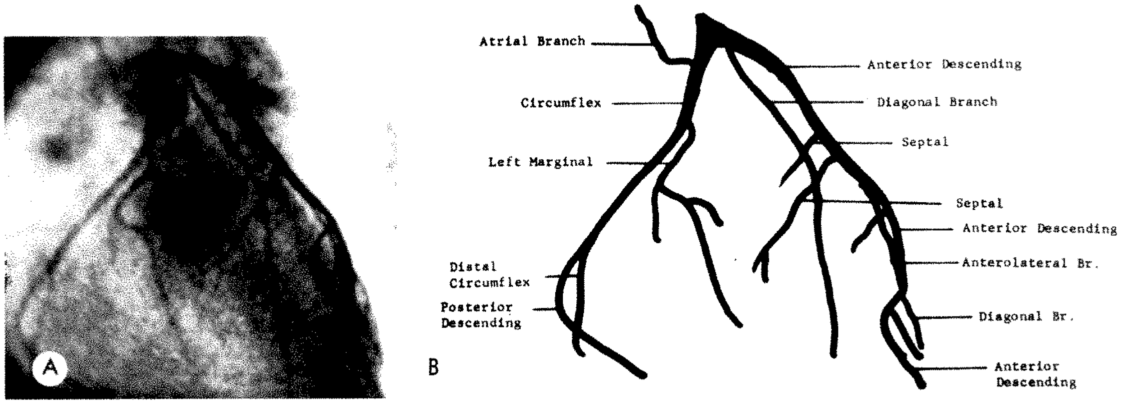

Fig. 10. (*A* and *B*) The left coronary artery of the same patient as in Figure 9, *A* and *B* in the right anterior oblique projection.

riogram, then it is reasonable to conclude that a branch is missing.

For instance, if a large circumflex artery appears to terminate in a small anterolateral branch at the obtuse margin, the only diagonal branch is of moderate size and arises from the anterior descending artery not more than 2 cm. to the left of the crux, then we can presume that there was originally a large left marginal branch which was not present at the time of the arteriogram.

Old myocardial infarcts require little arterial supply. Infarcts must not be confused with large areas of viable but ischemic myocardium. Fortunately, infarcts large enough to significantly affect the amount of myocardium remaining in a region of the left ventricle seem to be easily detectable on the electrocardiogram, and allowance can be made in estimating the amount of myocardium depending on the available arterial supply. Lack of movement or paradoxical motion of a portion of the cardiac border is often seen in appropriate projections when large infarcts are present. Clues can also be obtained by careful study of the pattern of movement of arteries in various portions of the heart during the cardiac cycle.

SUMMARY

Detailed information on the roentgenographic anatomy of the human coronary arterial system was worked out through study of anatomic literature, 3 dimensional vinylite corrosion casts of human coronary arteries, and cine arteriograms taken with selective injection of the right and left coronary arteries by the Sones technique.

The left anterior oblique projection was found best for the right coronary artery and its branches, the left main, distal anterior descending artery, the anterior descending branches, the proximal circumflex artery, and some of the proximal circumflex branches.

The right anterior oblique projection is also necessary because it is the only view that shows the proximal anterior descending and septal arteries well. It is often helpful for the midportion of the circumflex artery and its branches.

The posteroanterior projection is less satisfactory and cannot be considered an adequate substitute for either oblique projection. It is sometimes helpful as a supplement and has been recently included in routine coronary arteriography.

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CORONARY ARTERY CALCIFICATION*

ITS INCIDENCE AND SIGNIFICANCE IN PATIENTS OVER FORTY YEARS OF AGE

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CALCIFICATION of the coronary arteries is a common autopsy finding and is generally present in 80 to 90 per cent of postmortem studies performed in people over 40 years of age. Despite this common pathologic finding, roentgenographic identification of calcified coronary arteries in living patients has been a relatively rare event. The first reported case was in 1927 by Lenk,[6] who noted calcification of the left coronary artery on a posteroanterior roentgenogram of the chest in a 61 year old male with a left ventricular aneurysm. Other subsequent reports reflected the futility of identifying calcium in the coronary arteries with roentgenographic techniques. Sosman,[8] for example, in 1943 reported a total of 42 cases seen on fluoroscopy over a 10 year period. And in 1950, Habbe and Wright,[4] examining known cardiac patients over 40 years of age for a period of 2½ years with multiple high speed spot roentgenograms plus kymography, could identify calcified coronary arteries in 28 patients, representing an incidence of only 3 per cent of the cases studied. Thus, the evaluation of coronary artery disease still rested primarily on indirect evidence as manifested by clinical signs and electrocardiographic changes.

Yet, in 1959 a roentgenographic study of autopsied hearts by Blankenhorn and Stern[2] revealed an 80 per cent incidence of calcification of the coronary arteries and over one-half of the densities measured greater than 3 mm. The roentgenographic detection of coronary artery calcification took on an even greater significance when Beadenkopf *et al.*,[1] in 1964, reported their findings in a study of 904 consecutive autopsies. Their results indicated that as the number of coronary arteries with calcification increased, coronary artery wall thickness increased. Further, subjects without myocardial infarction showed a tendency for fewer arteries to be calcified, whereas those with myocardial infarction showed a tendency for more arteries to be calcified. If one assumes that calcification preceded the clinical appearance of coronary artery disease, then roentgenographic identification of calcium in coronary arteries might serve as a good indicator of potential ischemic heart disease.

Prior to the advent of image intensification, the lack of roentgenographic identification in living patients was attributed to overlying structures such as spine, ribs and calcified cartilage, to scattered radiation, and, most importantly, to the fact that one was trying to identify a small opaque particle on a rapidly moving structure. With the advent of image intensification and cineroentgenography, however, several of these limitations became negligible. Lesions as small as 2 mm. could now be resolved and rapidly moving densities were captured on cine-film which later might be reviewed for the presence of small densities. Further, all this was accomplished with reasonable radiation exposure.

Several investigators quickly appreciated the value of cineroentgenographic studies of intracardiac calcification. For example, in 1960, Jorgens, Blank and Wilcox[5] and in 1961 Edling[3] reported on its use in detecting the presence of intracardiac calcifications. Likewise, in 1961, Lieber and Jorgens[7] re-

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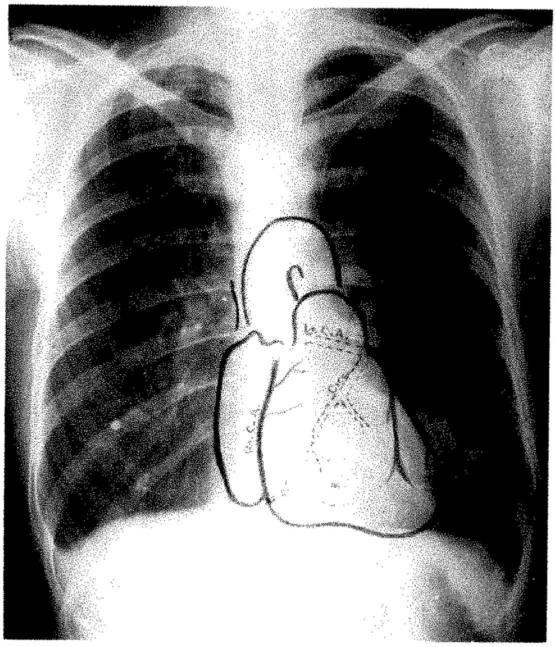

Fig. 1. Posteroanterior roentgenogram demonstrating the location of the right coronary artery and the left coronary artery and its branches.

ported a correlative study of coronary artery calcification and clinical disease. In both a control and cardiac group, they cited a 25 per cent incidence of calcification of coronary arteries in patients over the age of 40 years.

When cineroentgenography was introduced at the University of Vermont College of Medicine in 1961, it soon became apparent that a significant new dimension had become available as calcified coronary arteries were now more readily appreciated than they had previously been on conventional fluoroscopy. As this finding was recognized with increasing frequency, several questions arose. The first obvious question was, "How often can calcified coronaries be identified utilizing this new unit?" In order to answer this and other subsequent questions, the present study was carried out.

METHOD AND MATERIAL

From Januray 1, 1963 through July 1, 1964, all patients over the age of 40 years, male and female, inpatient and outpatient, who were studied on a 9 inch Philips Ringstand with remote television monitoring also had a cineroentgenographic survey of the heart. The patients included in the study were undergoing a variety of examinations, including barium swallow and upper gastrointestinal examinations, chest studies and heart fluoroscopy.

The 75 mm. overframing lens, resulting approximately in a 5 inch field rather than the 35.5 mm. conventional lens on a 16 mm. Arriflex Camera, was routinely employed as it was determined that the slight loss in image sharpness was more than compensated by the magnification effect produced by overframing. Since resolution on the cine-recording was greater than the resolution on the remote television monitor, final evaluation was deferred until the cineroentgenograms were reviewed. In several instances, the presence of coronary calcification was not noted on the remote television monitor but was later seen on the cine-recording. Factors utilized averaged about 75 kv. at 14 ma. Du Pont Cine Ray Film was used and developed in a Fisher Processall at a rate of 26 inches/min. in Kodak X-ray Developer. The patients were examined upright in the posteroanterior, right anterior oblique and left anterior oblique positions. The lateral view was not used in this study as it was felt that these 3 positions would yield all the information necessary to determine the presence of coronary artery calcification.

In the posteroanterior roentgenogram, (Fig. 1) the right coronary is somewhat difficult to visualize as it parallels the right heart border and tends to lie over the spine. The left is more easily seen, being located in the upper left border where it branches into the anterior descending and circumflex branches.

In the right anterior oblique roentgenogram (Fig. 2), the left coronary is located in the upper left border of the heart where it divides into the circumflex artery posteriorly and its anterior descending branch which continues along the upper left heart border laterally. In this view the right coronary lies further posteriorly and somewhat lower and ends inferiorly in a rather typical sling-like fashion along the inferior heart border.

The left anterior oblique roentgenogram (Fig. 3) was found to be the most informative and reliable. In this view the left coronary artery is seen in the upper left heart border where it branches laterally as the circumflex artery and anteriorly to form the anterior descending artery. The right coronary artery parallels the right border, thus presenting both arteries in opposite profile. Recordings were made in all three positions, utilizing cine-runs that lasted about 20 seconds.

RESULTS AND DISCUSSION

Utilizing this technique, 1,097 satisfactory cineroentgenographic examinations of the heart were performed. The group consisted of 522 males and 575 females. The presence of coronary artery calcification was then tabulated into one of three categories; right, left, and bilateral (Table 1).

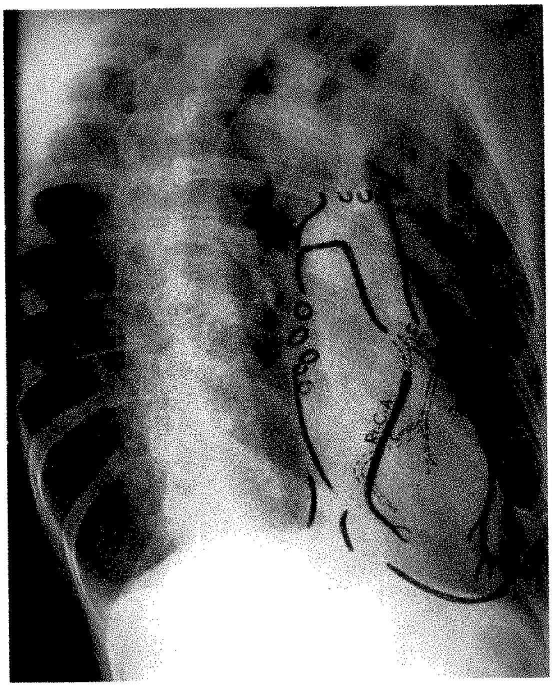

Fig. 2. Right anterior oblique roentgenogram demonstrating the location of the right coronary artery and the left coronary artery and its branches.

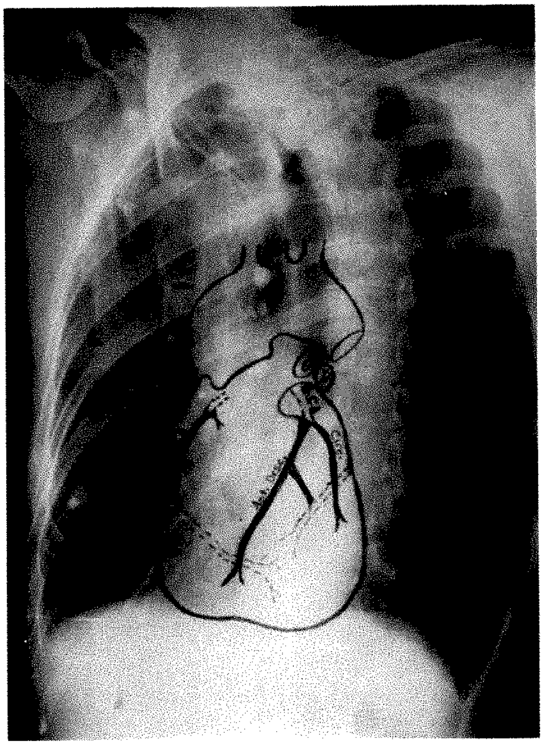

Fig. 3. Left anterior oblique roentgenogram demonstrating the location of the right coronary artery and the left coronary artery and its branches.

Calcification of the right coronary artery only was a rare finding, occurring in only 3 per cent of the males and in only 1 per cent of the females, or 2 per cent in the entire group. Calcification of the left coronary artery only, on the other hand, was relatively common, being observed in 12 per cent of the males and 7 per cent of the females with an over-all incidence of 9 per cent. Bilateral coronary artery calcification was observed in 4 per cent of both males and females. The total figures indicated that calcification occurred in 165 patients resulting in an over-all incidence of 15 per cent.

This figure falls considerably short of the 80 per cent incidence discovered in autopsy specimens. The discrepancy is probably due to intrinsic limitations of the examinations. Many calcifications are undoubtedly too small to be seen with the degree of resolution now available. In addition, calcifications in the heart inside the thorax are likely to be obscured by a thick chest wall, obesity or cardiomegaly. Thin emphysematous

TABLE I

INCIDENCE OF CORONARY ARTERY CALCIFICATION IN PATIENTS OVER THE AGE OF 40 YEARS

	Right		Left		Bilateral		Total	
	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
Males 522	15	3	63	12	20	4	98	19
Females 575	6	1	40	7	21	4	67	12
Total 1,097	21	2	103	9	41	4	165	15

patients proved to be easy patients to study, whereas thick obese patients with a large heart proved to be more difficult problems. Finally, overlying disease in the chest or pleura may also obscure coronary calcification. All these factors would tend to lower the incidence of recognition of calcified coronary arteries.

It should also be noted that our incidence of 15 per cent falls considerably below that of 25 per cent noted by Lieber and Jorgens. Two additional factors may help explain this. First, their groups were composed almost exclusively of males in a Veterans Administration hospital, whereas our material is from a general hospital population of males and females. Breaking our total figure of 165 cases down according to sex distribution resulted in the following: of 522 males in the study, 98 had identifiable coronary calcification, an incidence of 19 per cent, a figure more closely approaching those of other investigators. On the other hand, only 67 of 575 females, or 12 per cent, had visible calcified coronary arteries. Thus coronary artery calcifications are more common in males by a ratio slightly greater than 3:2, the discrepancy appearing to be related primarily to a higher incidence of calcification of the left coronary artery in males.

A second factor which may explain the discrepancy between our findings and those of other investigators in a study of this type is that of the limitations of the examiner. Lack of appreciation of the location or nature of coronary artery calcification may cause one to overlook a small fleck of calcification recorded on the film. This has frequently been observed while reviewing chest cine studies with the resident staff. It was also noted that the ease of recognition of coronary calcifications improved as the study progressed, suggesting that there is improved recognition as one becomes more familiar with the problem. Further, one may omit a cine run in one of the positions outlined or poorly position the patient while searching for coronary artery calcification and perhaps miss a small fleck of calcification which might have otherwise been identified. Finally, it is quite possible that the arbitrary decision to eliminate the lateral position in the protocol may have resulted in failure to identify additional cases with calcifications which might have been observed only in this position. Recognizing these limitations and realizing that they could only result in a lowering of the incidence of calcified coronary arteries, it was felt that the answer to the first question was as follows: utilizing this technique one could identify calcification in at least 15 per cent of patients over 40 years of age, and that it was more commonly seen in males in a ratio slightly greater than 3:2.

The relationship of the presence of calcification to age was next determined (Table II). As expected, there was a progressive increase in the incidence of calcified coronary arteries in each decade ranging from a low of about 4 per cent in the 40–49 year age group to a high of 29 per cent in the 80–89 year age group. The highest incidence, 35

Table II

AGE DISTRIBUTION OF CORONARY ARTERY CALCIFICATION

Age	Males		Females		Total	
	No.	Per Cent	No.	Per Cent	No.	Per Cent
(yr.)						
40–49	5	4	5	3	10	4
50–59	26	17	12	7	38	12
60–69	35	26	22	16	57	21
70–79	29	35	15	18	44	26
80–89	3	21	12	32	15	29
90–99	0	0	1	33	1	33

per cent, occurred in the 70–79 year old male group. The patients in the 90–99 year age group were too few to be considered as significant.

The next question that arose was what significance could one attach to the presence of calcification with respect to coronary artery disease. To answer this, the clinical charts of patients in this study were reviewed for the presence of the following information: history of angina, or electrocardiographic evidence of ischemia, or old or recent myocardial infarction. Utilizing these clinical criteria, 397 or 36 per cent of this group were determined to have some form of coronary artery disease. Of these, 85 or 21 per cent had calcification of the coronary arteries and 312 or 79 per cent had no evidence of coronary artery calcification (Table III). Thus it appeared that coronary artery disease frequently was present in the absence of calcified coronary arteries. The absence of coronary calcification, therefore, is not a reliable method of excluding coronary artery disease. In this respect, it cannot replace information obtained from clinical evaluation and electrocardiographic examination of patients with suspected heart disease.

Table III

INCIDENCE OF CALCIFIED CORONARY ARTERIES IN 397 PATIENTS WITH CLINICAL EVIDENCE OF CORONARY ARTERY DISEASE

	No.	Per Cent
With Calcification	85	21
Without Calcification	312	79

More interesting, however, was the evaluation next carried out on the calcified and noncalcified group (Table IV). Of the calcified group of 165, a total of 85 or 52 per cent had clinical coronary artery disease. On the other hand, 312 of 932, or only 33 per cent of the noncalcified group had clinical evidence of coronary artery disease. This finding indicated that there definitely was a higher incidence of clinically identified coronary artery disease among those with calcified coronary arteries than in

Table IV

INCIDENCE OF CORONARY ARTERY DISEASE IN PATIENTS WITH AND WITHOUT CORONARY ARTERY CALCIFICATION

Sex	Calcified Coronary Arteries	No. with Coronary Artery Disease	Per Cent	Noncalcified Coronary Arteries	No. with Coronary Artery Disease	Per Cent
Males	98	53	54	424	140	33
Females	67	32	48	508	172	34
Total	165	85	52	932	312	33

TABLE V

AGE DISTRIBUTION OF CORONARY ARTERY DISEASE IN PATIENTS WITH AND WITHOUT CORONARY ARTERY CALCIFICATION

Age (yr.)	Males				Females				Total			
	Calcified		Noncalcified		Calcified		Noncalcified		Calcified		Noncalcified	
	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
40–49	3	60	16	12	2	40	24	17	5	50	40	15
50–59	14	54	34	27	6	50	42	27	20	53	76	27
60–69	18	51	53	52	8	36	47	41	26	46	100	46
70–79	16	56	32	58	8	53	40	59	24	55	72	59
80–89	2	67	5	45	8	67	17	68	10	67	22	61
90–99	0	0	0	0	—	—	2	100	—	—	2	100

those without calcified coronary arteries. These findings were equally distributed between males and females.

Also of interest was the incidence of clinically identified coronary artery disease in the calcified and noncalcified groups when broken down according to decades (Table v). In the 40–49 and 50–59 year old groups, there was a far greater incidence of coronary artery disease in the calcified group. For example: 60 per cent of males in their forties with calcified coronary arteries had identified coronary artery disease, whereas only 12 per cent of a similar group without calcification had evidence of coronary artery disease; 54 per cent of males in their fifties with calcified coronary arteries had evidence of coronary artery disease, whereas only 27 per cent of a similar group without calcified coronary arteries had coronary artery disease. Similar findings were also noted in females where 40 per cent of those in their forties and 50 per cent of those in their fifties with coronary calcification had coronary artery disease, whereas only 17 per cent of those in their forties and 27 per cent of those in their fifties without calcification had coronary artery disease.

TABLE VI

DISTRIBUTION OF CORONARY ARTERY DISEASE IN PATIENTS OVER 60 AND UNDER 60 YEARS OF AGE WITH AND WITHOUT CALCIFIED CORONARY ARTERIES

	With Calcification		Without Calcification	
	No.	Per Cent	No.	Per Cent
Under 60				
Male	17	55	50	19
Female	8	47	66	22
Total	25	52	116	21
Over 60				
Male	36	54	90	54
Female	24	48	106	51
Total	60	51	196	52

Once over 60 years of age, the incidence of heart disease in the calcified and noncalcified group was more nearly equal. By tabulating the cases of heart disease in patients under and over 60 years old (Table vi), it was noted that the total incidence of coronary artery disease in patients under 60 years with coronary calcification was strikingly higher than in the noncalcified group. For example, the over-all incidence of coronary artery disease in patients under 60 years old with coronary artery calcification was 52 per cent, whereas it was found in only 21 per cent of those without calcification. This difference was apparent in both males and females. Once over 60 years of age, the relationship was no longer apparent and coronary artery disease appeared to be more equally dis-

TABLE VII

RELATIONSHIP OF CORONARY ARTERY DISEASE TO LOCATION AND NUMBER OF CORONARY ARTERY CALCIFICATION

	None Calcified		Right Only		Left Only		Right and Left	
	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
Male	140	33	7	47	32	51	14	70
Female	172	34	3	50	16	40	13	62
Total	312	33	10	48	48	47	27	66

tributed between patients with and without calcification.

A final study was carried out in an attempt to relate coronary artery disease to the location and extent of coronary artery calcification (Table VII). As expected, there appeared to be a direct relationship between the number of arteries calcified and the presence of clinically apparent disease. In those patients with no calcification, the incidence was 33 per cent. In those with either the left or right coronary artery calcified, the incidence increased to about 48 per cent. In those with bilateral calcification, the incidence climbed to 66 per cent. With slight variations, this rising incidence held for both the male and female populations. It is reasonable to assume that if the distribution of calcification in the left coronary artery had been broken down into those of the left circumflex and anterior descending artery, an even stronger relationship would have been discovered between coronary artery disease and calcification of all three vessels.

The results of this study indicate that a distinct relationship exists between coronary artery calcification and coronary artery disease. It must be emphasized that the level of information obtained with this technique cannot approach the nature of information obtained from coronary angiography where precise evaluation of coronary flow and obstruction can be determined. However, coronary angiographic studies require complex equipment, are time consuming and are not without hazard. This technique on the other hand is simple, rapid and innocuous. Its greatest value may be in the asymptomatic patient with a normal electrocardiogram, for in these patients the presence of coronary artery calcification may prove to be a good indicator of potential ischemic heart disease. Since cineroentgenographic detection of calcification in the coronary arteries may be considered as an alarm signal for high risk coronary artery disease in an asymptomatic patient, it is recommended that a search for coronary artery calcification with image intensification be carried out as a routine screening procedure on all fluoroscopic examinations.

SUMMARY AND CONCLUSIONS

1. The incidence and significance of coronary artery calcification were determined on the basis of television monitored cineroentgenographic studies of hearts of 1,097 patients over the age of 40 years.

2. As the study progressed, it became increasingly apparent that the identification of coronary artery calcification is related not only to intrinsic factors such as the quality of the television picture and the cine-reproduction, but also to the skill and experience of the observer.

3. The over-all incidence of coronary artery calcification in patients over 40 years of age is 15 per cent. It is more common in males by a ratio slightly greater than 3:2.

4. The incidence of calcification of the coronary arteries increases with increasing age.

5. Coronary artery disease is frequently

present in the absence of identifiable coronary artery calcification. Thus, absence of recognizable coronary artery calcification is unreliable in excluding the presence of coronary artery disease. However, the incidence of coronary artery disease in patients with coronary artery calcification is greater than in those without calcification. Further, when broken down by decades there is a strikingly high incidence of coronary artery disease in young patients with coronary artery calcification. In patients under the age of 60 years, the incidence of coronary artery disease in those with calcified coronary arteries is 52 per cent, whereas in those without calcification it is only 21 per cent. In patients over the age of 60 years, the incidence of coronary artery disease in the calcified and noncalcified group is nearly equal.

6. A relationship exists between the incidence of coronary artery disease and the number of coronary arteries calcified. In patients with noncalcified coronary arteries, the incidence of coronary artery disease is 33 per cent, with one artery calcified 48 per cent, and with both arteries calcified 66 per cent.

7. Cineroentgenographic detection of calcification of the coronary artery may be considered as an alarm signal of potential ischemic heart disease in asymptomatic patients with a normal electrocardiogram. It is, therefore, recommended that a search for coronary artery calcification with image intensification be carried out as a routine screening procedure in all fluoroscopic examinations.

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RIGHT AORTIC ARCH: PLAIN FILM DIAGNOSIS AND SIGNIFICANCE*

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IN THIS paper we discuss the anatomic types of right aortic arch in terms of their relative incidences, their significance, and how each can be recognized by simple roentgenographic methods. Our discussion is based on data obtained from reviewing clinical histories, roentgenograms, surgical reports, and autopsy specimens in 298 cases of right aortic arch with levocardia, representing 10 years' experience (1954 to 1964) at the Mayo Clinic.

If the anatomic type of right aortic arch could be established clearly from the autopsy specimen, the angiocardiogram, or the surgical report, the case was designated as proved. Roentgenograms of the thorax and esophagus were reviewed in the proved cases and, from this review, it became apparent that the anatomic type of right aortic arch could be diagnosed from these roentgenograms with a high degree of accuracy. In those cases in which neither autopsy, angiocardiography, nor operation was done but in which the thoracic and esophageal roentgenograms demonstrated the type of right aortic arch, the designation "qualified proved" was assigned.

After separation of the proved and qualified proved cases, a group of undesignated cases remained in which the anatomic type could be inferred with moderately good accuracy because study of the proved cases demonstrated (and the literature also indicated) that there is a high degree of correlation between the anatomic type of right aortic arch and the presence or absence of certain types of congenital heart disease. Therefore, a group designated as "presumed" could be established on the basis of the cardiac findings. Thirteen cases of right aortic arch could not be classified as to type.

CLASSIFICATION

In a recently published monograph,[5] a classification of malformations of the aortic arch was proposed. The embryologic error responsible for each of a wide variety of aortic arch anomalies was determined, and then these errors were reduced into individual component parts and systematically recombined in all plausible combinations. Most of the anomalies in the classification thus formulated have been described in the literature, but some are, as yet, hypothetical. The right aortic arch segment of that classification was followed in this study.

There are 3 major types of right aortic arch: (1) with mirror-image branching of the major arteries (that is, the mirror image of the branching present in a normal left aortic arch) (Fig. 1, *A*, *B* and *C*), (2) with aberrant left subclavian artery (Fig. 2, *A*, *B* and *C*), and (3) with left subclavian artery no longer connected (Fig. 3, *A*, *B* and *C*). Each of these three major types of right aortic arch theoretically may have either a left ductus arteriosus,* a right ductus arteriosus, or bilateral ducti arteriosi.

ANATOMIC AND ROENTGENOGRAPHIC FEATURES

RIGHT AORTIC ARCH WITH MIRROR-IMAGE BRANCHING

In specimens of right aortic arch with mirror-image branching, the aorta passes

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* The term "ductus arteriosus" is used throughout this paper to denote either a patent ductus arteriosus or its remnant, a ligamentum arteriosum.

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From the Mayo Clinic and Mayo Foundation: Section of Roentgenology (Drs. Stewart and Kincaid) and of Experimental and Anatomic Pathology (Dr. Titus).

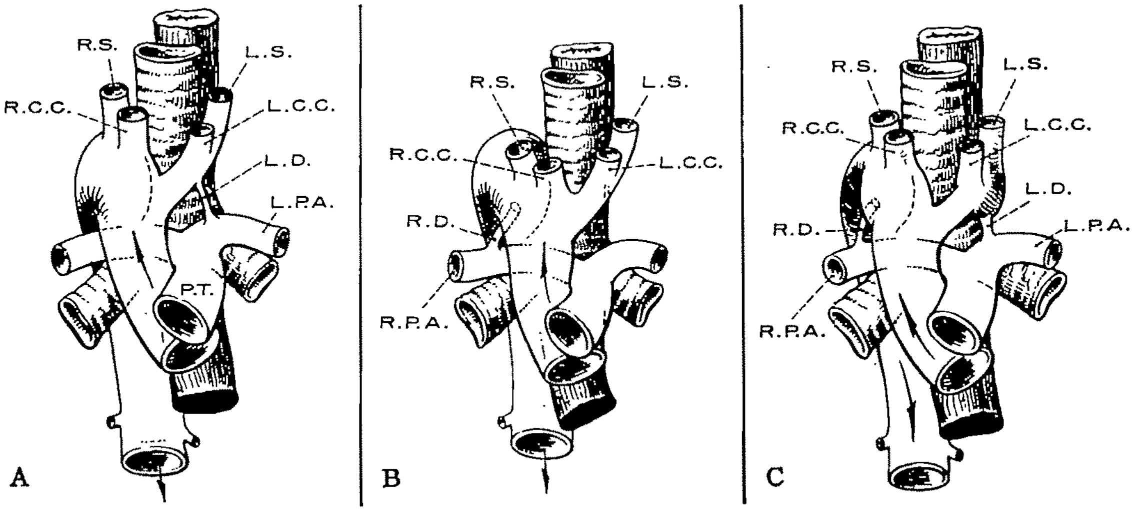

Fig. 1. Right aortic arch with mirror-image branching, as viewed from the front. The anomaly with (*A*) left ductus arteriosus, (*B*) with right ductus arteriosus, and (*C*) with both ducti present. Key to abbreviations for all figures: A.A., ascending aorta; A.L.S., aberrant left subclavian artery; D.A., descending aorta; E., esophagus; In.A., innominate artery; L.C.C., left common carotid; L.D., left ductus; L.P.A., left pulmonary artery; L.S., left subclavian artery; P.T., pulmonary trunk; R.C.C., right common carotid; R.D., right ductus; R.P.A., right pulmonary artery; R.S., right subclavian artery; TR., trachea; L.D.A.R., left dorsal aortic root; P.L.D., patent left ductus. (Reproduced with permission.[6])

to the right of the trachea and descends toward the normal aortic hiatus entirely on the right side of the esophagus (Fig. 4, *A* and *B*). Arising from the aorta are the left innominate, right common carotid, and right subclavian arteries, in that order from front to back. Among the autopsy specimens in which ductal tissue could be identified adequately, most specimens of right aortic arch with mirror-image branching had a left ductus, a few had a right ductus, and in 1 specimen both ducti were clearly present.

The location of the ductus arteriosus in

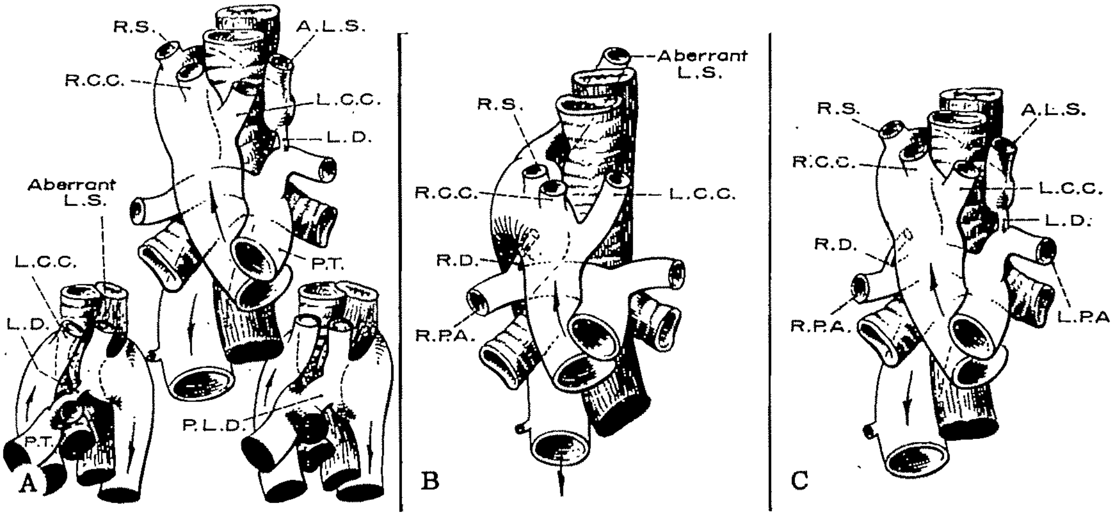

Fig. 2. Right aortic arch with aberrant left subclavian artery as viewed from the front. (*A*) With left ductus arteriosus (*insets* show view from left side with closed [*lower left*] and patent [*lower right*] ductus). (*B*) With a right ductus arteriosus. (*C*) With both ducti present. (Reproduced with permission.[6])

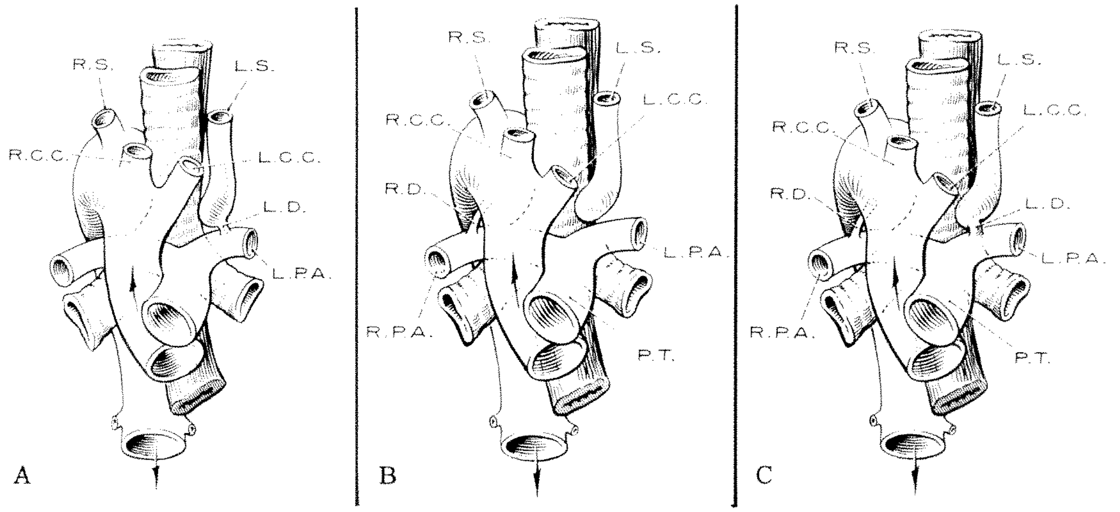

Fig. 3. Right aortic arch with isolation of the left subclavian artery from the aorta as viewed from the front. (*A*) With left ductus arteriosus. (*B*) With right ductus arteriosus. (*C*) With both ducti. (*A* and *C* reproduced with permission.[5])

this anomaly does not alter its fundamental configuration,* significance, or roentgenographic appearance and, for purposes of simplification, these 3 variations in the position of the ductus arteriosus will hereafter be ignored and the composite term "right aortic arch with mirror-image branching" will be used.

A right aortic arch with mirror-image branching can be recognized from the

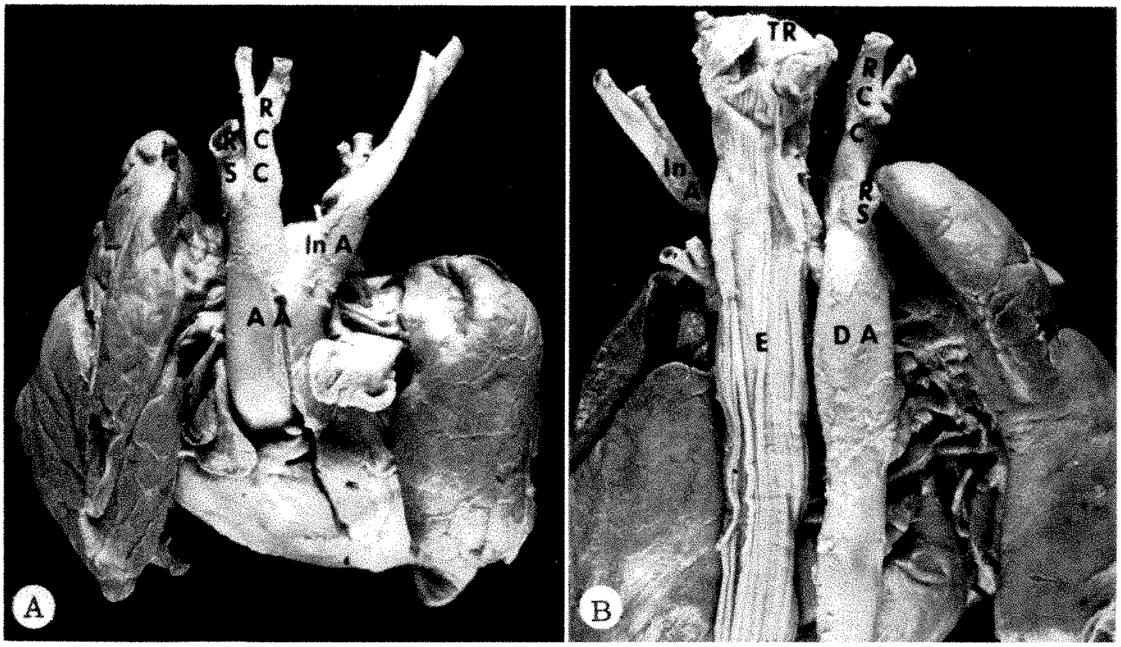

Fig. 4. Heart and lungs of a 7 month old boy who had tetralogy of Fallot and right aortic arch with mirror-image branching. (*A*) Viewed from the front. (*B*) Viewed from behind. The esophagus has been opened. Since the aorta does not cross behind the esophagus, there is no compression of the esophagus from behind. In this specimen, there was a left ductus arteriosus.

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* An exception is the extremely rare instance in which the left ductus connects to the upper descending aorta instead of to the left innominate artery, forming a vascular ring. An example of this variation was not encountered in this series.

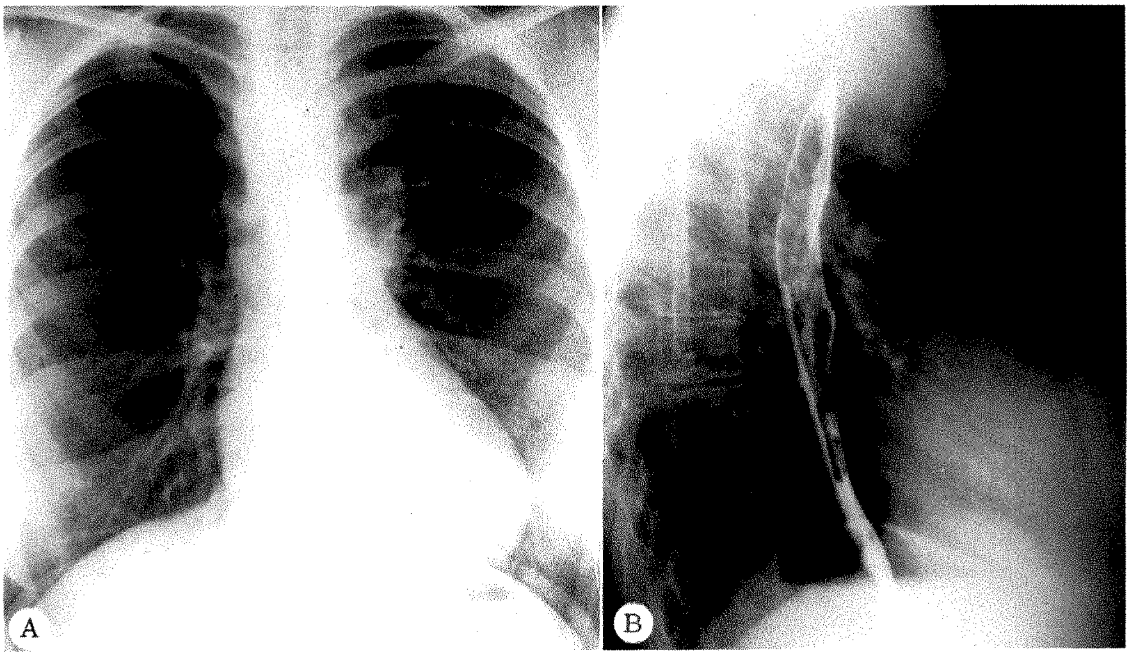

FIG. 5. These roentgenograms of a 20 year old woman with tetralogy of Fallot and an atrial septal defect are diagnostic of right aortic arch with mirror-image branching. (*A*) The aortic arch is distinctly on the right and compresses the right side of the trachea. The upper descending aorta is faintly discernible to the right of the spinal column. There is a conspicuous vacancy to the left of the trachea in the usual location of the aortic arch. (*B*) Lateral view of the esophagus, which is outlined by barium, demonstrating that there is no retroesophageal compression.

thoracic roentgenogram and the lateral view of the barium-filled esophagus (Fig. 5, *A* and *B*). On the thoracic roentgenogram the right-sided arch indents the right side of the trachea or pushes the trachea slightly to the left. The upper descending aorta can usually be seen through the mediastinal tissues to the right of the spinal column. There is no vascular component on the left side of the trachea. The cardiac silhouette is rarely normal since the great majority of these patients have congenital heart disease. The lateral view of the barium-filled esophagus is normal because this anomaly does not have a retroesophageal component.

RIGHT AORTIC ARCH WITH ABERRANT LEFT SUBCLAVIAN ARTERY

The major branches of the aorta are, from front to back, the left common carotid, right common carotid, right subclavian, and aberrant left subclavian arteries. In contrast to right aortic arch with mirror-image branching, the configuration and significance of this anomaly is considerably altered by the location of the ductus arteriosus. If the ductus is on the left, the aberrant left subclavian artery is anchored down toward the left pulmonary artery; the trachea and esophagus are completely encased by the ascending aorta and pulmonary artery anteriorly, the right aortic arch on the right, the upper descending aorta posteriorly, and the aberrant left subclavian artery and left ductus arteriosus on the left (Fig. 6, *A*–*D*). The proximal portion of the aberrant left subclavian artery is bulbous because, embryologically, it is formed from what would have been the posterior portion of the left aortic arch. The retroesophageal vascular component of this anomaly is quite large as it is formed by the confluence of the upper descending aorta, right arch, and bulbous proximal portion of the aberrant left subclavian artery. This anomaly resembles a double aortic arch.

When a right aortic arch with aberrant

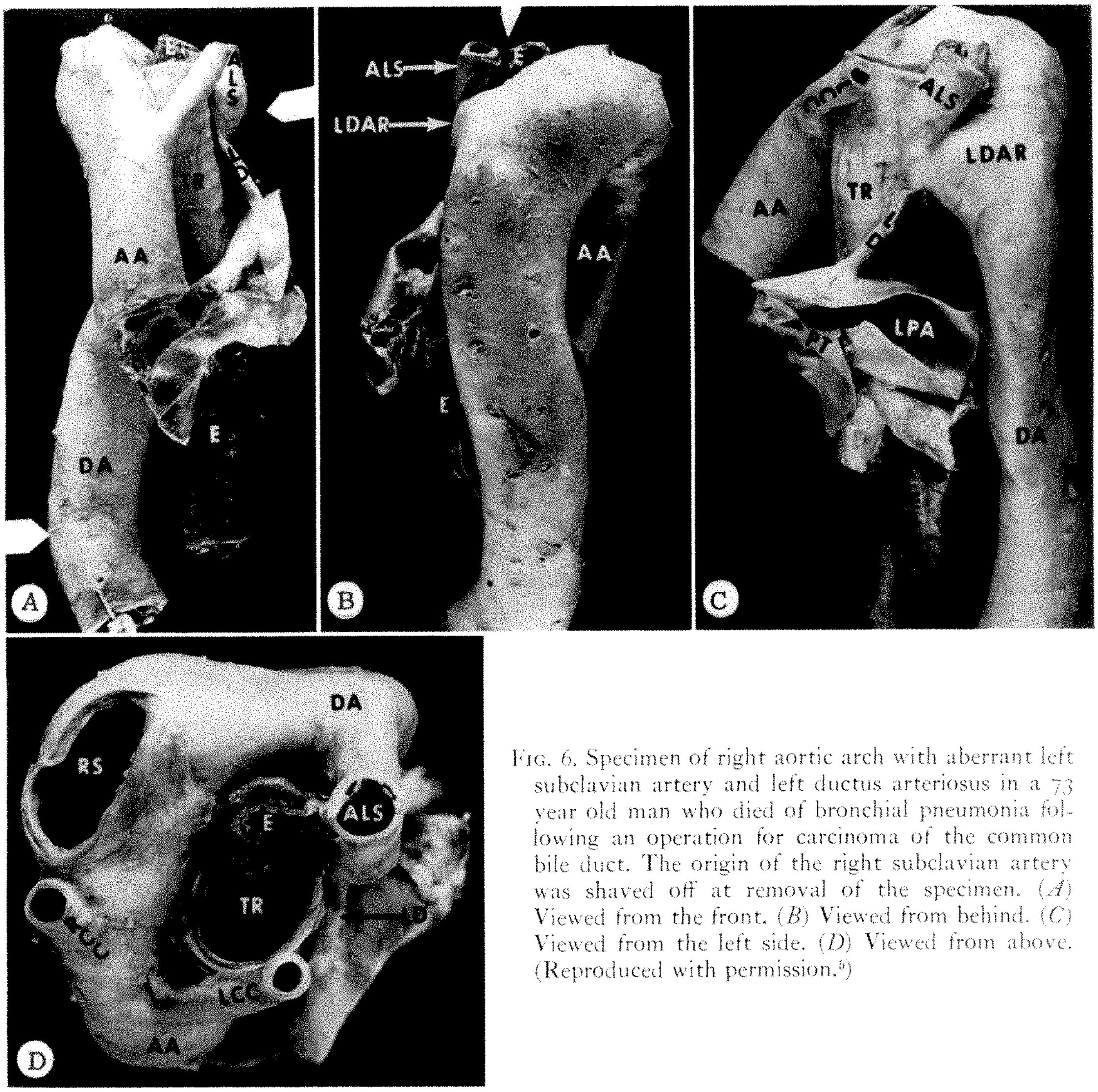

Fig. 6. Specimen of right aortic arch with aberrant left subclavian artery and left ductus arteriosus in a 73 year old man who died of bronchial pneumonia following an operation for carcinoma of the common bile duct. The origin of the right subclavian artery was shaved off at removal of the specimen. (*A*) Viewed from the front. (*B*) Viewed from behind. (*C*) Viewed from the left side. (*D*) Viewed from above. (Reproduced with permission.[5])

left subclavian artery has a right ductus arteriosus, the aberrant subclavian artery is at an angle of approximately 70° from horizontal as it passes behind the esophagus to reach the left arm, a complete vascular ring is not present, and the trachea and esophagus are not significantly compressed. Although this anomaly is the exact mirror image of the very common left aortic arch with aberrant right subclavian artery and thus might be expected to be common, it is rare.

Right aortic arch with aberrant left subclavian artery and bilateral ducti arteriosi is as yet hypothetical since no such specimen has been reported in man. It would be a complete vascular ring having the same configuration as a right aortic arch with aberrant left subclavian artery and left ductus arteriosus.

Right aortic arch with aberrant left subclavian artery nearly always has a left ductus arteriosus, and this anomaly usually produces rather typical roentgenographic findings (Fig. 7, *A*, *B* and *C*). The ascending aorta arches far to the right, producing a vascular shadow projecting to the right of the mediastinum. After passing over the right main-stem bronchus adjacent to the right side of the trachea, the

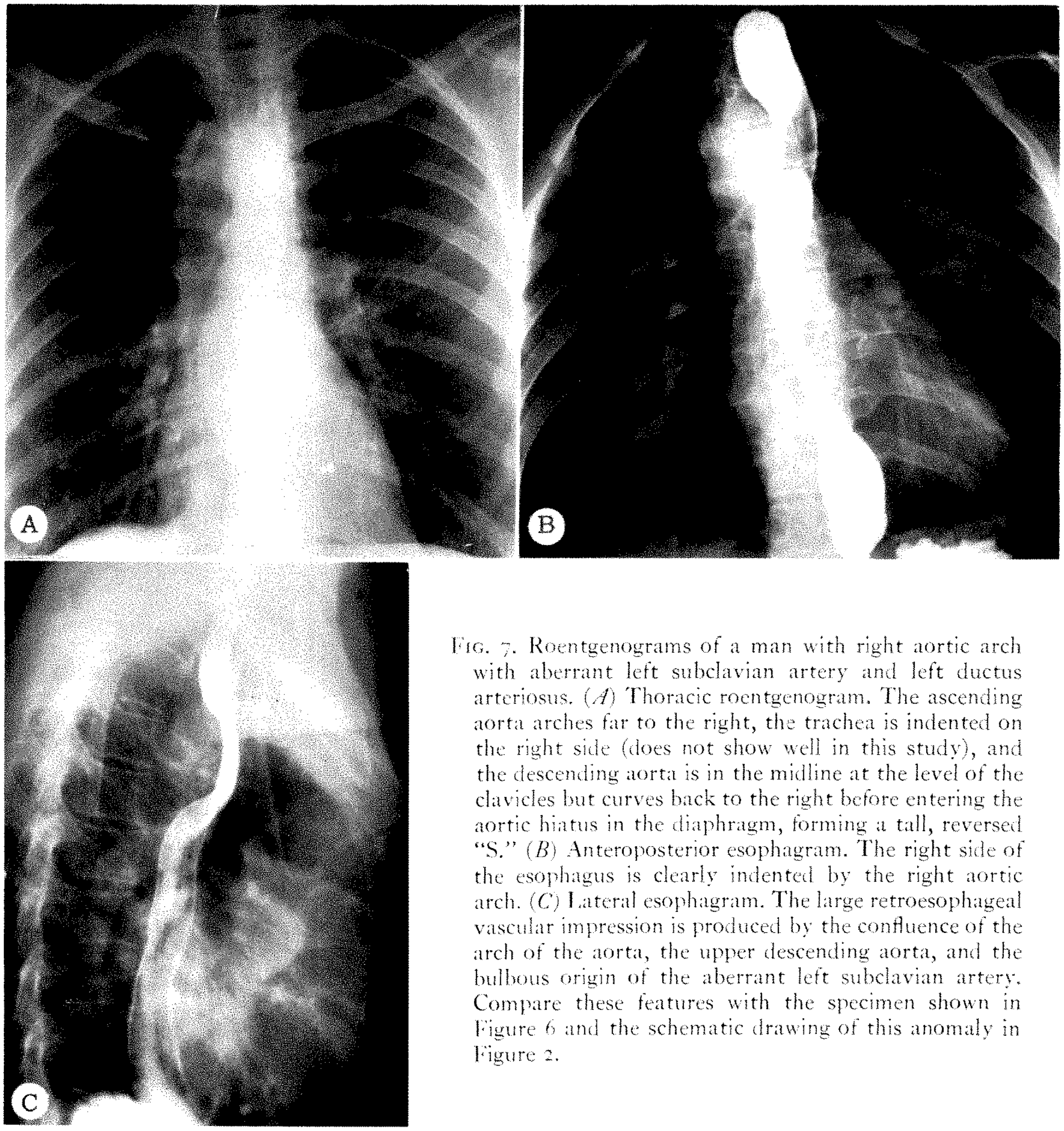

Fig. 7. Roentgenograms of a man with right aortic arch with aberrant left subclavian artery and left ductus arteriosus. (*A*) Thoracic roentgenogram. The ascending aorta arches far to the right, the trachea is indented on the right side (does not show well in this study), and the descending aorta is in the midline at the level of the clavicles but curves back to the right before entering the aortic hiatus in the diaphragm, forming a tall, reversed "S." (*B*) Anteroposterior esophagram. The right side of the esophagus is clearly indented by the right aortic arch. (*C*) Lateral esophagram. The large retroesophageal vascular impression is produced by the confluence of the arch of the aorta, the upper descending aorta, and the bulbous origin of the aberrant left subclavian artery. Compare these features with the specimen shown in Figure 6 and the schematic drawing of this anomaly in Figure 2.

arch of the aorta joins the upper descending aorta where the latter gives origin to the aberrant left subclavian artery behind the esophagus. The origin of the aberrant left subclavian artery is bulbous and often this diverticulum-like structure can be seen on the thoracic roentgenogram, projecting to the left of the trachea at a slightly lower level than the arch of the right-sided aorta. The descending aorta then usually descends to the right of the spinal column before entering the normal aortic hiatus. As the aorta undergoes torsion in older patients, it often assumes the shape of a tall, thin, reversed "S" with the midportion of the descending aorta crossing to the right of the midline before it curves back to enter the normal aortic hiatus.

In contrast to patients with right aortic arch with mirror-image branching, the cardiac silhouette in patients with right aortic arch with aberrant left subclavian artery and left ductus arteriosus is usually normal because this type of anomaly is seldom associated with congenital heart disease.

This anomaly can also be recognized rather accurately from the esophagram. On an anteroposterior roentgenogram of the barium-filled esophagus, the right side of the esophagus is indented by the right aortic arch as in other types of right aortic arch. There is a conspicuous absence of the similar indentation on the left side of the esophagus that is usually seen in patients with double aortic arch. A lateral view of the barium-filled esophagus shows a large, concave impression produced by the retroesophageal vascular component of this anomaly. In fact, the retroesophageal component of this type of right aortic arch is so large that both the esophagus and trachea often are displaced anteriorly at this level. These thoracic and esophageal findings are quite different from those seen in patients with right aortic arch with mirror-image branching.

In infants, exact roentgenologic diagnosis may be more difficult because the aorta is often badly obscured by a large thymus. If the aorta can be recognized as being right sided either on the thoracic roentgenogram or on fluoroscopy of the mediastinum and esophagus, a lateral study of the barium-filled esophagus will distinguish right aortic arch with mirror-image branching from right aortic arch with aberrant left subclavian artery and left ductus arteriosus. If right aortic arch with mirror-image branching is present, there is no retroesophageal impression. If a large retroesophageal defect is present, it may be impossible to differentiate between double aortic arch and right aortic arch with aberrant left subclavian artery and left ductus arteriosus, even with angiography.[5]

Right aortic arch with aberrant left subclavian artery and right ductus arteriosus is extremely rare. It can easily be recognized and distinguished from the other types of right aortic arch by its appearance on the esophageal studies (Fig. 8, *A–D*). On the anteroposterior and lateral roentgenograms of the barium-filled esophagus, there is a shallow retroesophageal vascular groove which angles upward from right to left at approximately 70° from the horizontal. This esophageal impression is the exact mirror image of an impression produced by the common aberrant right subclavian artery arising from the normal left aortic arch. The shallowness and the angle of the vascular groove distinguish this anomaly from right aortic arch with aberrant left subclavian artery and left ductus arteriosus (Fig. 8*C*).

RIGHT AORTIC ARCH WITH ISOLATION OF LEFT SUBCLAVIAN ARTERY

The anomaly of right aortic arch with the left subclavian artery isolated from the aorta is rare. This anomaly is not a vascular ring but seems to occur in association with congenital anomalies of the heart. Figure 9, *A* and *B*, shows the specimen from a case of right aortic arch with isolation of the left subclavian artery and left ductus arteriosus.

The findings on roentgenograms of the thorax and barium-filled esophagus are probably indistinguishable from those noted in patients with right aortic arch with mirror-image branching. Thus, there is a right-sided aortic arch without posterior indentation of the esophagus but with an abnormal cardiac silhouette.

Since this anomaly is so rare, we usually ignore it as a possibility when interpreting the roentgenograms in patients with right aortic arch. Nevertheless, the radiologist and the cardiovascular surgeon should be aware of this anomaly.

DISCUSSION

Incidence. Of the 298 cases of right aortic arch, 285 could be classified into the anatomic types defined herein (Table 1). Ninety-eight per cent of the classified cases of right aortic arch had either mirror-image branching or aberrant left subclavian artery and left ductus arteriosus.

Although apparently contrary to the data of Table 1, it is our belief that right aortic arch with aberrant left subclavian

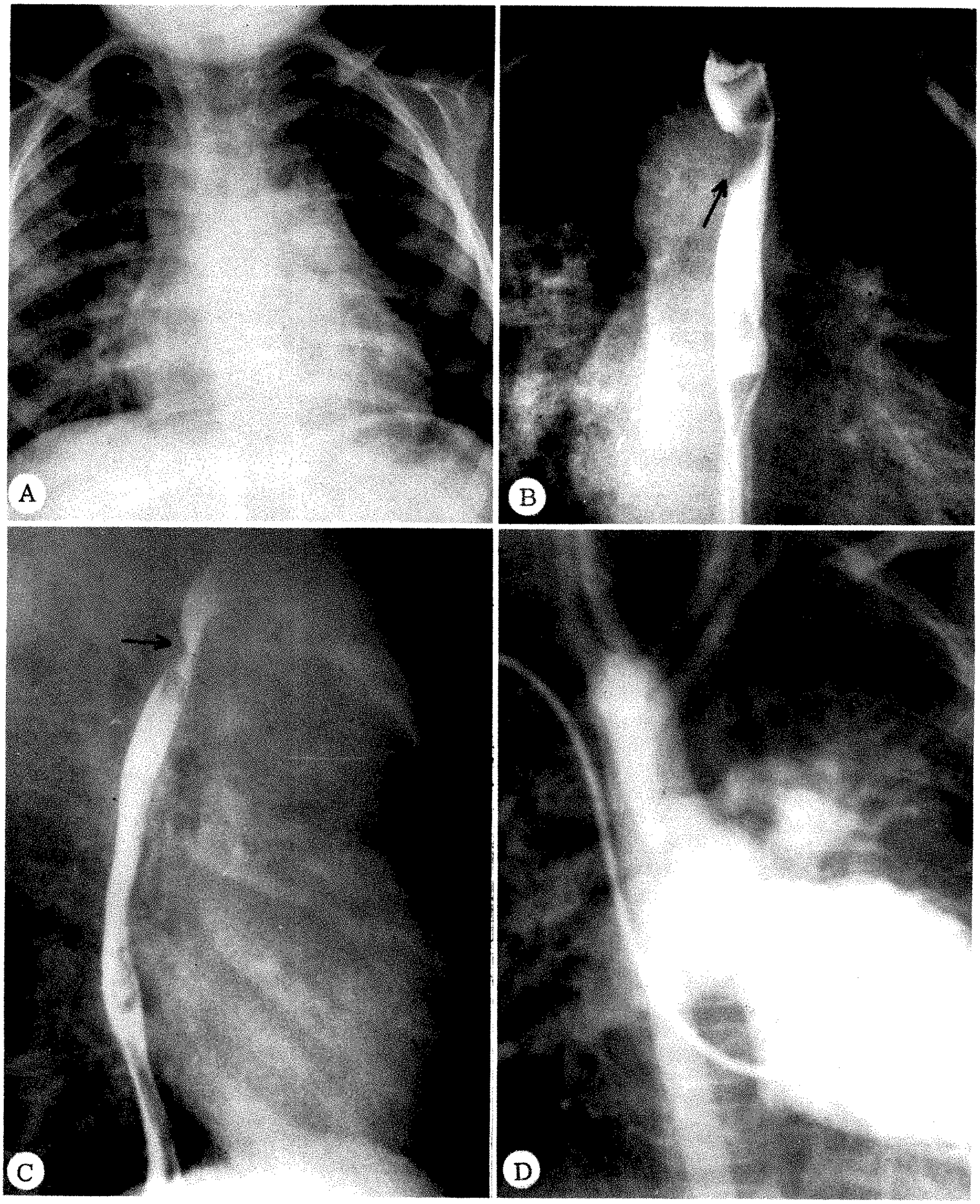

FIG. 8. Roentgenograms of a 2 year old girl who had no clinical evidence of esophageal or tracheal compression but did have congenital heart disease. (*A*) Cardiac enlargement and right-sided aortic arch. (*B*) Compression defect which angles upward from right to left across the column of barium at approximately 70° from horizontal (*arrow*) is produced by the aberrant left subclavian artery. (*C*) Lateral roentgenogram of the esophagus demonstrates a small retroesophageal defect (*arrow*). (*D*) Angiocardiogram suggesting tetralogy of Fallot and aortic configuration of a right aortic arch with aberrant left subclavian artery and right ductus arteriosus.

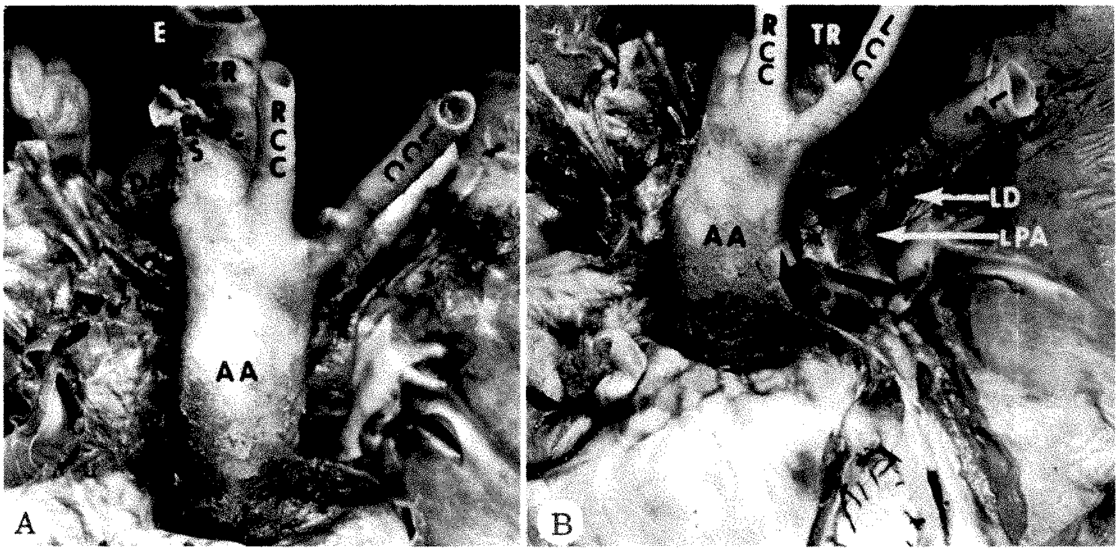

Fig. 9. Heart and lung of a 6 year old boy who had right aortic arch with isolation of left subclavian artery and patent left ductus arteriosus (patient died after operative repair of tetralogy of Fallot). (*A*) Viewed from the front. (*B*) Viewed from the left. The patent ductus arteriosus from which the left subclavian artery arises has a ligature around it. There is no innominate artery arising from the aorta since the left subclavian artery does not have a common origin with the left common carotid artery. (Reproduced with permission.[5])

artery and left ductus arteriosus is the most common type of right aortic arch. A series such as the one reported herein is weighted in favor of right aortic arch with mirror-image branching because (1) many patients were referred to this clinic for

Table I

RELATIVE INCIDENCE OF TYPES OF RIGHT AORTIC ARCH

Type	Proved (no.)	Qualified Proved (no.)	Presumed (no.)	Total	
				No.	Per Cent
Mirror-image branching Left ductus Right ductus Bilateral ducti	130	6	33	169	59.3
Aberrant left subclavian artery					
Left ductus	17	56	40	113	39.5
Right ductus	1	0	0	1	0.4
Bilateral ducti	0	0	0	0	—
Isolated left subclavian artery					
Left ductus	2	0	0	2	0.8
Right ductus	0	0	0	0	—
Bilateral ducti	0	0	0	0	—
Total classified	150	62	73	285	100
Indeterminate type of right arch				13	
Grand total				298	

TABLE II

TYPE OF CARDIOVASCULAR ANOMALY FOUND IN ASSOCIATION WITH RIGHT AORTIC ARCH

Type of Right Aortic Arch	Tetralogy of Fallot	Truncus	Transposition	ASD and/or VSD	Coarctation	Indeterminate Defect	Other*	Total
Mirror-image branching Left ductus Right ductus Bilateral ducti	147	4	2	1	—	—	12	166
Aberrant left subclavian artery								
Left ductus	9	—	—	3	1	—	—	13
Right ductus	1	—	—	—	—	—	—	1
Isolated left subclavian artery								
Left ductus	1	1	—	—	—	—	—	2
Indeterminate	—	7	—	4	—	2	—	13
Total	158	12	2	8	1	2	12	195

ASD = atrial septal defect; VSD = ventricular septal defect.

* These included one case each of: VSD, partial anomalous pulmonary venous connection; VSD, corrected transposition; tricuspid atresia, subpulmonary stenosis; single ventricle, pulmonary atresia, corrected transposition; single ventricle, mitral atresia, pulmonary stenosis; VSD, pulmonary atresia, situs inversus of abdominal viscera; cor biloculare, mitral atresia, pulmonary stenosis, anomalous pulmonary venous connection, situs inversus of viscera; cor biloculare, subpulmonary stenosis; double outlet right ventricle, pulmonary stenosis, atrioventricular canal; double outlet right ventricle, VSD; double outlet right ventricle, multiple other defects; agenesis of the left pulmonary artery, normal heart.

correction of congenital heart disease and, as will be shown, right aortic arch with mirror-image branching occurs almost exclusively in patients with congenital heart disease, and (2) patients with right aortic arch and mirror-image branching and congenital heart disease undergo frequent thoracic roentgenography and often angiocardiography, operation, and, occasionally, autopsy—all of which provide many opportunities for diagnosis of each case. On the other hand, most patients with right aortic arch with aberrant left subclavian artery and left ductus arteriosus do not have associated malformations of the heart and usually have had only 1 thoracic roentgenogram made; therefore, there is less chance for these cases to be diagnosed, recorded, and included in a study. It is our unsubstantiated impression that right aortic arch with aberrant left subclavian artery and left ductus arteriosus is probably 2 or 3 times more common in the general population than is right aortic arch with mirror-image branching.

Accuracy of Roentgenographic Diagnosis. Because 98 per cent of patients with right aortic arch have either mirror-image branching or aberrant left subclavian artery and left ductus arteriosus, and an additional small fraction have aberrant left subclavian artery and right ductus arteriosus, all of which are readily distinguishable on the thoracic roentgenogram and lateral studies of the barium-filled esophagus, the roentgenographic diagnosis of the exact type of right aortic arch can be made from these roentgenograms with a considerable degree of accuracy.

Association of Congenital Heart Disease with Right Aortic Arch. Among the patients with right aortic arch with mirror-image branching, 98 per cent had congenital heart disease and 2 per cent had normal

Table III

INCIDENCE AND TYPE OF RIGHT AORTIC ARCH IN TETRALOGY OF FALLOT, TRUNCUS ARTERIOSUS, AND TRANSPOSITION

Type of Right Aortic Arch		Tetralogy of Fallot	Truncus Arteriosus	Transposition
	Total No. of Cases Examined	555	25	80
Mirror-image branching Left ductus Right ductus Bilateral ducti		142 (93%)	4	2
Aberrant left subclavian artery				
Left ductus		9 (6%)	0	0
Right ductus		1 (0.5%)	0	0
Bilateral ducti		0	0	0
Isolated left subclavian artery				
Left ductus		1 (0.5%)	1	0
Right ductus		0	0	0
Bilateral ducti		0	0	0
Indeterminate		0	4	0
Total		153 (100%)	9	2
Per cent of total cases with right arch		28	36	2.5

hearts. Among those with right aortic arch with aberrant left subclavian artery and left ductus arteriosus, 12 per cent had congenital heart disease and 88 per cent had normal hearts. In all, 195 of the 298 patients with right aortic arch had congenital heart disease (Table II). Of the remaining 103, 100 had complete vascular rings formed by the combination of right aortic arch, aberrant left subclavian artery, and left ductus arteriosus. Thus, 99 per cent of our patients with right aortic arch had either congenital heart disease or an aortic vascular ring. As previously shown, right aortic arch with mirror-image branching can be distinguished roentgenographically from right aortic arch with aberrant left subclavian artery and left ductus arteriosus. Accurate diagnosis of the type of aortic arch, therefore, will lead to accurate assessment of the presence or absence of congenital heart disease or a vascular ring. Eighty-seven per cent (147 of 169) of the patients with right aortic arch with mirror-image branching had tetralogy of Fallot.

Association of Right Aortic Arch with Congenital Heart Disease. Table III summarizes the incidence of different types of right aortic arch associated with various forms of congenital heart disease. These groups of congenital heart disease represent cases which have been encountered at the Mayo Clinic during the last 10 years, and only cases which had been studied by angiocardiography, operation, or autopsy were included. Of these, only tetralogy of Fallot, truncus arteriosus, and transposition of the great vessels warrant presentation since the incidence of right aortic arch in other specific types of congenital heart disease was found to be almost nil. This study suggests that the incidence of coexistent right aortic arch is highest in truncus arteriosus.

Significance of Right Aortic Arch with Aberrant Left Subclavian Artery and Left

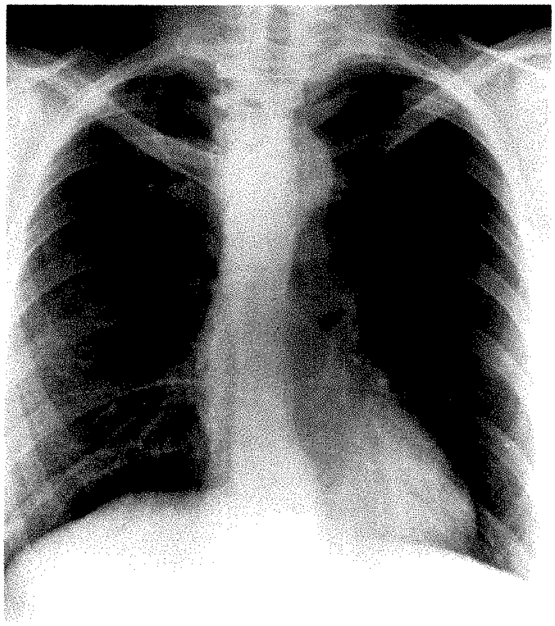

FIG. 10. Roentgenogram of a patient with right aortic arch with aberrant left subclavian artery and left ductus arteriosus. Patient underwent exploratory thoracotomy with the presumptive diagnosis of substernal goiter. There were no symptoms of tracheal or esophageal compression.

Ductus Arteriosus. Right aortic arch with aberrant left subclavian artery and left ductus arteriosus is a complete vascular ring. The critical period for patients with vascular rings is infancy and early childhood. Only 15 of our 113 patients with this anomaly were in the pediatric age group and of these, 3 had sufficient symptoms to warrant operative division of the ring. Because this anomaly is common and can cause considerable constriction of the trachea and esophagus, it is one of the three most important vascular rings of infancy and early childhood, the other two being double aortic arch and "anomalous left pulmonary artery."

Most patients with this anomaly survive infancy without operation and, in fact, the anomaly often does not cause symptoms. The 12 other children and 98 adults in this series had few or no symptoms of tracheal or esophageal compression at the time the anomaly was recognized.

If right aortic arch with aberrant left subclavian artery and left ductus arteriosus does not cause symptoms, it usually goes undetected until adult life when an abnormal mediastinal shadow is recognized on the thoracic roentgenogram. Not infrequently, and particularly in older individuals, as the aorta undergoes torsion and dilatation, this anomaly resembles a mediastinal tumor. Several patients in this series were thought to have mediastinal masses, on the basis of the original thoracic roentgenograms. Usually, the correct diagnosis was established by fluoroscopy but one patient included in the series (and one other patient since this series has been completed) underwent exploratory thoracotomy with a presumptive clinical diagnosis of substernal goiter (Fig. 10). Similar diagnostic problems have been reported by others.[1-4,6]

CONCLUSIONS

1. If a right aortic arch is present, there is a 99 per cent chance that the patient has either congenital heart disease or a vascular ring which completely encircles the trachea and esophagus.

2. Two main types of right aortic arch comprise 98 per cent of the cases of right aortic arch: (1) right aortic arch with branching of the major arteries, that is, a mirror image of the branching present in the normal left aortic arch; and (2) right aortic arch with an aberrant left subclavian artery and left ductus arteriosus. Of these 2, the latter is probably the most frequent type of right aortic arch in the general population.

3. These 2 types of right aortic arch are easily recognizable and distinguishable on thoracic roentgenograms and lateral roentgenograms of the barium-filled esophagus. These two procedures are now part of our routine examination in patients with suspected congenital heart disease.

4. When a right aortic arch with mirror-image branching is present, there is a 98 per cent chance that the patient has coexistent congenital heart disease and only a 2 per cent chance that the heart is normal. Of those with coexistent congenital heart

disease, the heart anomaly is tetralogy of Fallot in approximately 90 per cent; the remaining 10 per cent are a variety of different anomalies.

5. Right aortic arch with aberrant left subclavian artery and left ductus arteriosus is one of the important complete vascular rings which is capable of interfering with respiration and swallowing during early life, thereby occasionally necessitating operative division of the vascular ring. However, this anomaly usually does not significantly compress the trachea and esophagus, and, therefore, most often goes unnoticed until adult life when its peculiar appearance may mislead the radiologist into making the diagnosis of a mediastinal mass.

6. Only 12 per cent of patients with right aortic arch with aberrant left subclavian artery and left ductus arteriosus have associated congenital heart disease.

7. Twenty-eight per cent of 555 patients with tetralogy of Fallot had right aortic arch, and approximately 93 per cent of these were with mirror-image branching.

8. Thirty-six per cent of 25 patients with persistent common truncus arteriosus had right aortic arch.

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ROENTGENOLOGIC FINDINGS IN CONDITIONS OF THE ASCENDING AORTA SIMULATING AORTIC VALVULAR INSUFFICIENCY*

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THE roentgenologist dealing with cardiovascular disease is frequently confronted with roentgenograms of patients suspected of having aortic valvular insufficiency. In analyzing such cases it is important to determine (1) whether or not aortic insufficiency is the result of intrinsic disease of the aortic valve itself or (2) whether the fundamental disease process lies in the ascending aorta.

The purpose of the authors is to consolidate within one communication the roentgenologic and angiocardiographic findings of certain diseases of the ascending aorta responsible for abnormal run-off of blood through the aortic valve or through channels other than the aortic valve. These diseases fall into two major categories: (1) those causing secondary incompetence of the valve, thus allowing return of blood to the left ventricle through the orifice of the valve, and (2) those characterized by an abnormal communication between the ascending aorta, on one hand, and a cardiac chamber or a large mediastinal vessel, on the other.

The case material which forms the basis for this report is in the combined pathologic and roentgenologic collections of the University of Minnesota Hospitals and the Cardiovascular Registry of The Charles T. Miller Hospital. The pathologic findings of conditions of the ascending aorta causing aortic valvular insufficiency were reviewed earlier by one of us (R.S.E.).[9]

I. ESCAPE OF BLOOD THROUGH AORTIC VALVE

Conditions of the ascending aorta causing aortic valvular insufficiency are (A) dilatation of the ascending aorta, (B) tearing of the ascending aorta, or both occurring together.

Dilatation of the ascending aorta is the most common process and is a manifestation of medial weakness. The causes of medial weakness include (1) syphilitic aortitis, (2) rheumatoid spondylitis, (3) cystic medial necrosis as seen in Marfan's syndrome, idiopathic dilatation of the aorta and some other heritable disorders, and (4) advanced age (senile dilatation).

Tearing of the ascending aorta may cause (1) a "complete dissecting" aneurysm of the aorta, or (2) a site of localized aneurysmal dilatation, or so-called "incomplete dissecting" type of aneurysm.

From a roentgenographic viewpoint, the effects of aortic insufficiency upon the circulation are the same whether the abnormal flow results from intrinsic aortic valvular disease or from disease of the ascending aorta. In each process, the left ventricle becomes enlarged owing to the acceptance of an increased volume of blood during diastole. As will be shown in the following sections, it is usually the roentgenographic changes in the ascending aorta and general skeletal structure which serve to distinguish the various causes of aortic regurgitation. At times, differentiation is impossible without selective aortography.

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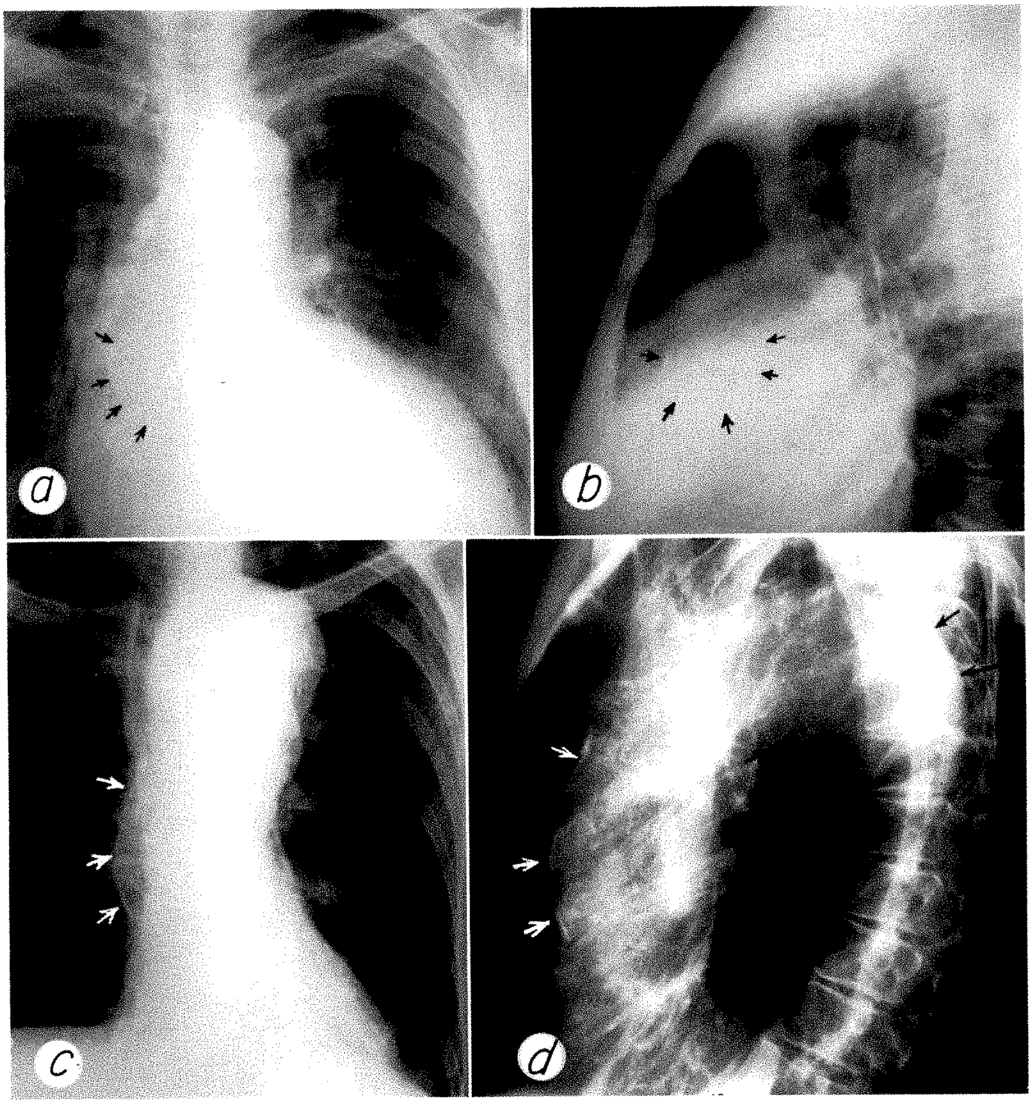

FIG. 1. Syphilitic aortitis. Thoracic roentgenograms in 2 cases. (*a*) Frontal and (*b*) lateral views of a 55 year old man. Each shows dense, linear calcifications (arrows) confined to the ascending aorta, a prominent ascending aorta and left ventricular enlargement. The lateral view shows calcification (arrows) of the aortic sinuses as well. (*c*) Frontal and (*d*) lateral views of a 60 year old man. Dense linear calcifications (arrows) confined to the ascending aorta. There is dilatation of the ascending aorta and an aneurysm of the arch (confirmed by thoracic aortography). The latter (black arrows) seen better in the lateral view. The cardiac silhouette is not enlarged.

A. DILATATION OF AORTA

1. *Syphilitic Aortitis.* This is the classic condition in which aortic valvular insufficiency results from aortic dilatation.[6]

With the exception of aortic dilatation, specific roentgenographic features do not appear until the disease process is advanced. The commonly associated intimal calcification of the ascending aorta and arch is the factor from which a strong suspicion of syphilitic aortitis may be entertained roentgenographically (Fig. 1, *a–d*). This calcification is usually a manifestation of atherosclerosis superimposed upon the underlying syphilitic process and not of syphilis *per se*.

In association with calcification of the ascending aorta, there usually is dilatation and elongation of the thoracic aorta. The calcification is manifested as thin deposits in the ascending aorta. In some instances the calcification may be seen in frontal view but is seen best in oblique and lateral views (Fig. 1, *a–d*). The root of the aorta may also show evidence of calcification, as may the descending thoracic aorta. Calcification of the thoracic aorta has been reported to occur in 40 per cent of cases of syphilitic aortitis.[27] There are usually no abnormal roentgenologic changes until the condition is advanced.

While syphilitic aortitis is by far the most common condition underlying calcification of the ascending aorta, we have observed the same finding in other conditions involving this portion of the aorta. These include idiopathic dilatation, atherosclerosis and localized aneurysms.

When aortic valvular insufficiency is present, the left ventricle shows considerable enlargement. Not infrequently, the arch is larger than the heart (Fig. 1*c*).

The degree of valvular incompetence may be accompanied by pronounced dilatation of the aortic sinuses (Valsalva). The latter feature is best demonstrated by selective thoracic aortography (Fig. 2, *a* and *b*).

2. *Rheumatoid Spondylitis.* Rheumatoid spondylitis may be responsible for aortic insufficiency and pathologically the lesion is difficult to distinguish from syphilitic aortitis.[3,5] In rheumatoid spondylitis the lesions tend to be confined to the ascending aorta yet they spare the aortic sinuses whereas in syphilitic aortitis the lesions may involve the entire thoracic and the aortic sinuses as well.

Roentgenologically, the diagnosis of aortic incompetence on the basis of rheumatoid spondylitis may be suggested when the ascending aorta is dilated and is associated with skeletal changes of rheumatoid spondylitis.

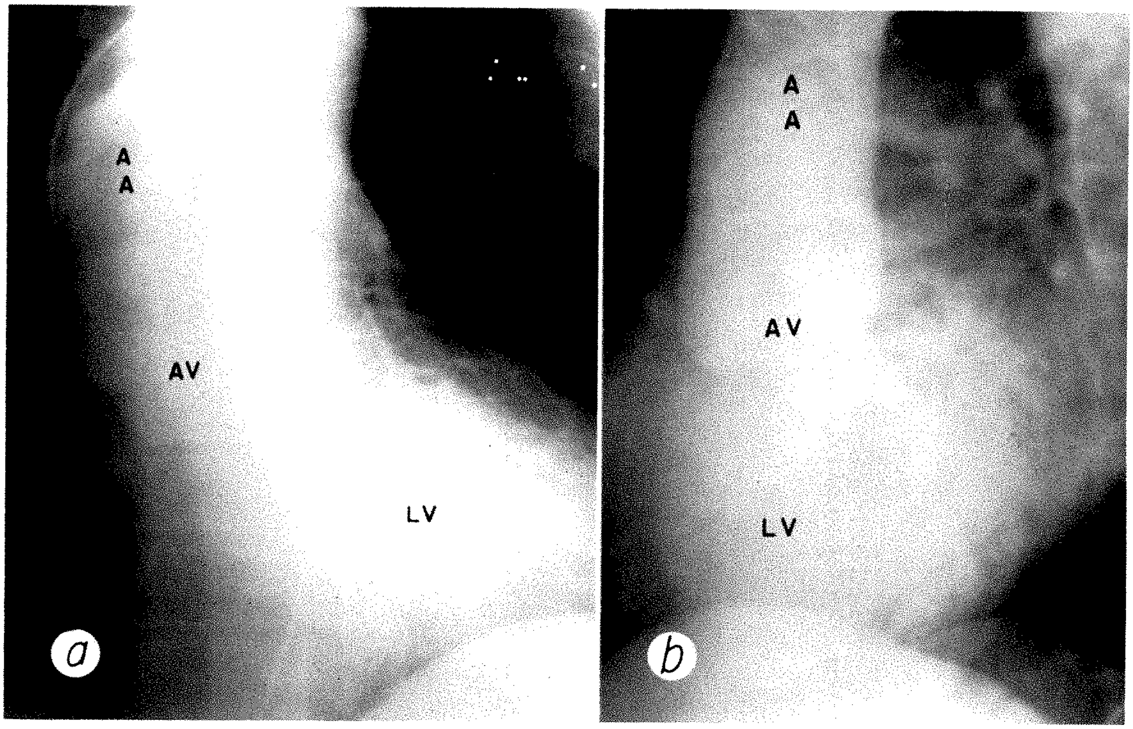

FIG. 2. Syphilitic aortitis. (*a* and *b*) Frontal and lateral selective thoracic aortograms. Same patient as shown in Figure 1, *a* and *b*. There is fusiform dilatation of the ascending aorta (AA) with dense opacification of the left ventricle (LV), indicating severe aortic valvular incompetence. AV = aortic valve.

3. *Cystic Medial Necrosis of the Aorta.* The congenital disturbance of connective tissue which is manifested in Marfan's syndrome is often associated with aortic medial disease.[20,24,30,31] The basic lesion is extensive fragmentation, interruption and rarefaction of elastic fibers of the media with mucoid accumulations. Other than dilatation of the ascending aorta leading to aortic insufficiency, complications of the aortic medial disease include the formation of saccular aneurysms or of dissecting aneurysms. Other stigmata frequently present are arachnodactyly, luxation or subluxation of the optic lenses, kyphoscoliosis, pectus excavatum and high palate.

Cystic medial necrosis of the aorta has been described in several heritable disorders. This process may occur in gargoylism,[28] Ehlers-Danlos syndrome,[10] and osteogenesis imperfecta.[4] When a patient with one of the foregoing heritable disorders displays roentgenologic signs of aortic valvular insufficiency, the diagnosis of cystic medial necrosis of the ascending aorta is permissible.

It is important to realize that cystic medial necrosis of the aorta occurs in varying degrees among people with apparently normal body habitus. In such patients, involvement of the aorta has been termed idiopathic dilatation of the aorta, a condition considered to represent a *forme fruste* of Marfan's syndrome, although the latter point is difficult to prove or disprove.[32]

In each condition, the roentgenographic findings of the heart and great vessels are identical and will be presented together.

The data obtained from roentgenograms and cardiac roentgenoscopy usually are those of classic aortic valvular insufficiency. These are (1) varying degrees of prominence of the ascending aorta, (2) left ventricular enlargement, and (3) increased pulsations of the entire thoracic aorta (Fig. 3, *a–d;* and 4, *a–d*). In cystic medial necrosis, the aorta shows considerably more prominence than in examples of intrinsic aortic valvular insufficiency (Fig. 3, *a–d*). The prominence may be so great that the lateral views not infrequently show the ascending aorta occupying the entire retrosternal space above the anterior cardiac border (Fig. 3*d*).

In some cases, dilatation of the ascending aorta is confined predominantly to the region of the aortic valve and the proximal part of the ascending aorta, each of which is situated within the cardiovascular silhouette. As a result, the frontal roentgenogram does not reveal the true extent of the aortic dilatation (Fig. 5, *a* and *b;* and 6, *a–d*).[29] In such cases, dilatation of the ascending aorta is usually recognizable in the left anterior oblique and/or lateral views. An additional roentgenographic sign may be of help. The aortic sinuses and the suprabulbar portion of the ascending aorta may be so dilated as to yield the effect of a mass over the middorsal spine. Penetrated grid roentgenograms are essential to define the phenomenon (Fig. 3*a;* and 6*b*).

The most striking angiocardiographic feature is marked and symmetric dilatation of the ascending aorta restricted to the ascending portion and including the aortic sinuses (Fig. 3 through 6). Characteristically, the wall is smooth and uniform. In addition, generally there is pronounced dilatation of the aortic sinuses. The dilatation is usually so pronounced that the aortic sinuses are poorly defined and may be described as "effaced." Typically, the ascending aorta and aortic sinuses in the lateral view resemble in shape a Florence flask (Fig. 3*d*). As the aorta is traced distally its caliber changes abruptly to normal (Fig. 3 through 6).

The degree of aortic incompetence is judged best by selective aortography (Fig. 3 through 6), without which the true diameter of the dilated portion of the ascending aorta cannot accurately be evaluated (Fig. 6, *c* and *d*).

4. *Senile Dilatation of the Ascending Aorta.* In elderly individuals the aorta regularly is dilated. This is a commonly recog-

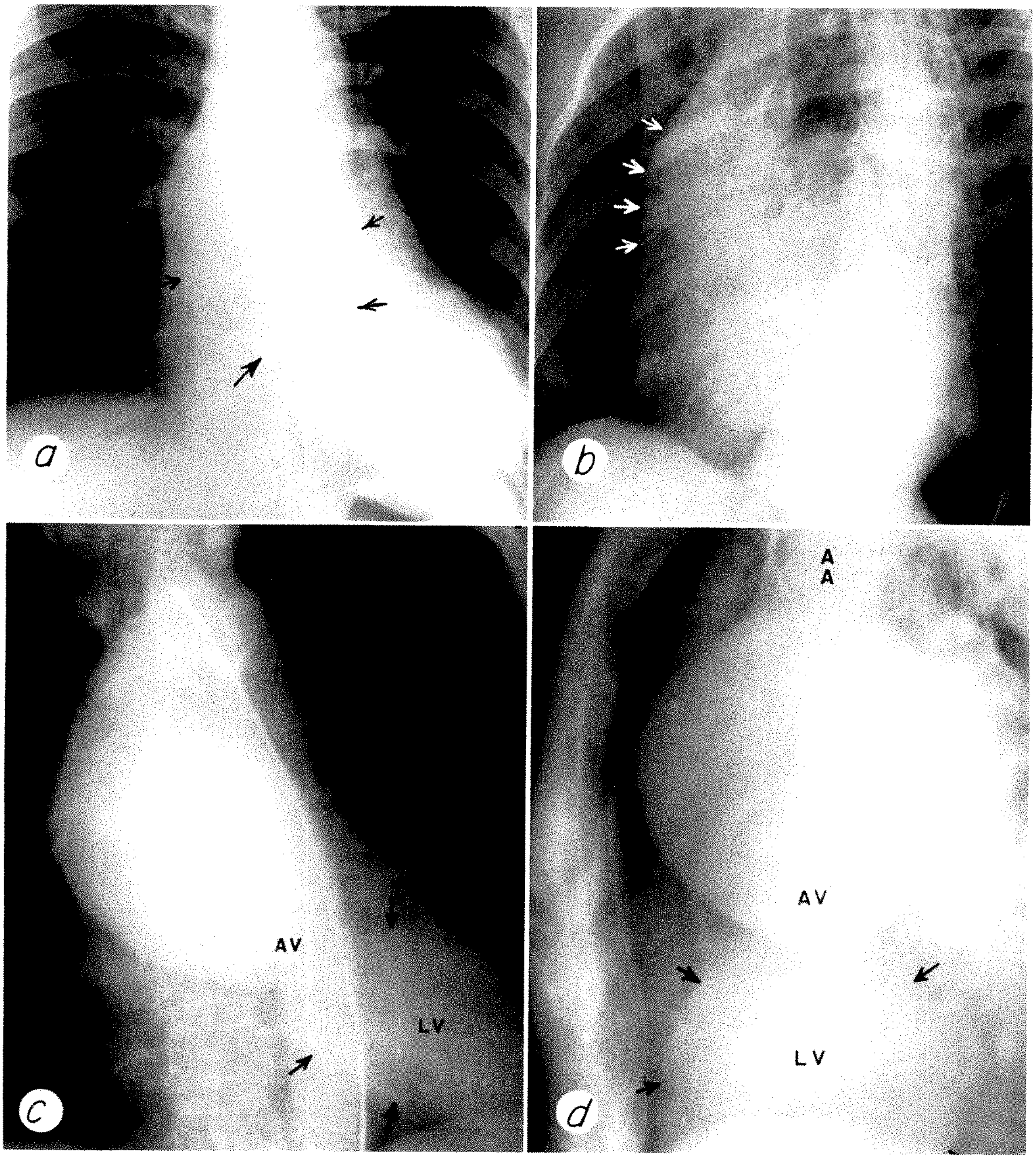

FIG. 3. Idiopathic dilatation of the ascending aorta in a 24 year old man with aortic valvular regurgitation. Abbreviations as in Figure 2. (*a*) Frontal and (*b*) left anterior oblique views, respectively, showing pronounced enlargement of the ascending aorta and of the left ventricle. (*c*) Frontal and (*d*) lateral thoracic aortograms. There is pronounced dilatation of the upper two-thirds of the ascending aorta and of the aortic sinuses. Abrupt change between the normal and abnormal portions. The dilatation is associated with severe aortic valvular regurgitation.

nized but poorly understood phenomenon which is to be charged to progressive weakness of the media. It is not secondary to atherosclerosis, although this lesion may coexist when the aorta is dilated in aged subjects.

Though some degree of aortic elongation and widening of the ascending aorta is

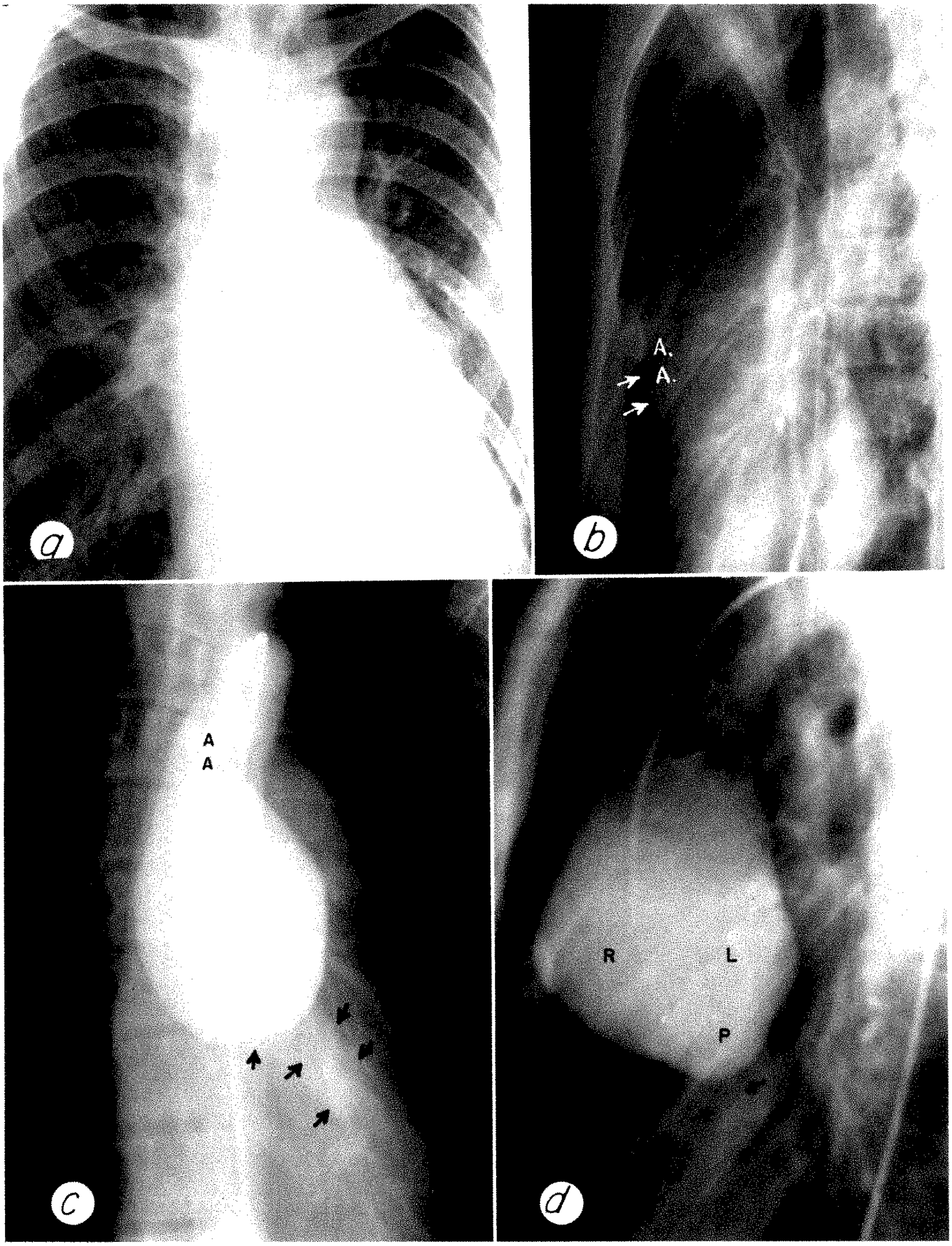

Fig. 4. Marfan's syndrome with aortic valvular regurgitation in a 19 year old man. Abbreviations as in Figure 2. (*a*) Frontal thoracic roentgenogram. There is evidence of only moderate prominence of the ascending aorta. (*b*) Left anterior oblique view shows considerable dilatation of the ascending aorta (A.A.). (*c*) Frontal and (*d*) lateral thoracic aortograms. There is marked dilatation of the lower portion of the ascending aorta, of the aortic root and of the aortic sinuses. Abrupt change in caliber between the dilated portion and the normal portion of the ascending aorta. In addition, there is aortic valvular regurgitation (arrows). R = right; P = posterior and L = left aortic sinuses, respectively.

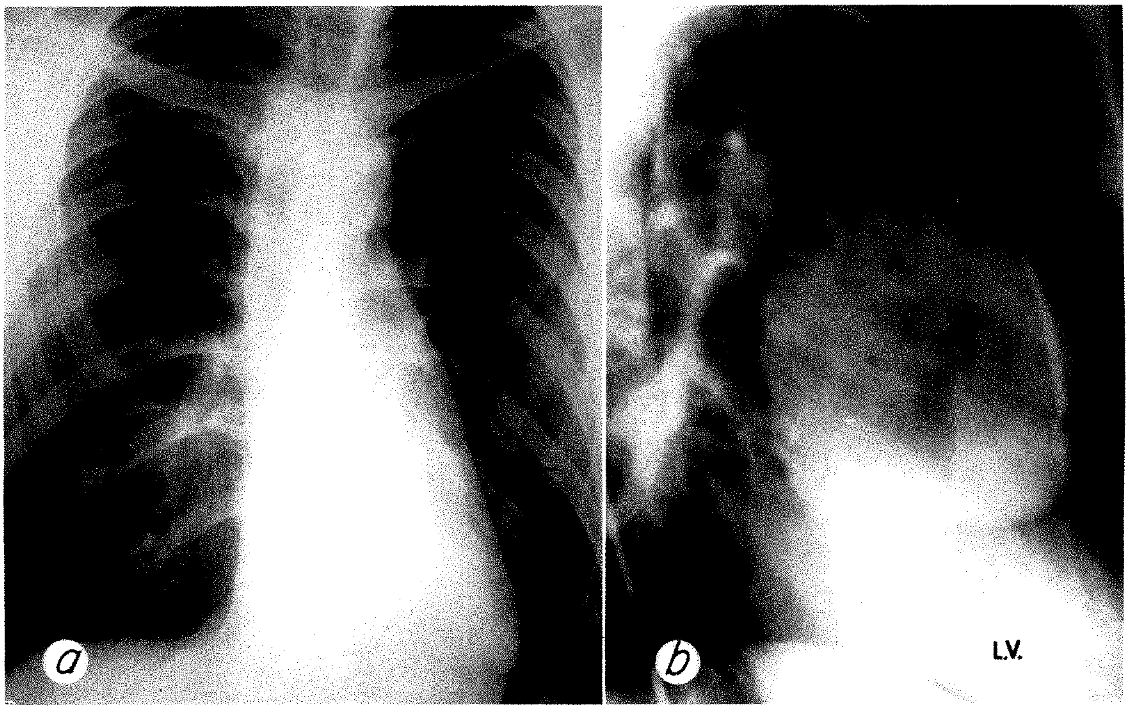

FIG. 5. Marfan's syndrome in a 19 year old man with aortic valvular regurgitation. Abbreviations as in Figure 2. (*a*) Frontal thoracic roentgenogram fails to show dilatation of the ascending aorta. (*b*) Thoracic aortogram shows typical aneurysmal dilatation of the lower ascending aorta and of the aortic sinuses. Aortic valvular regurgitation is also evident.

universally seen in patients of advanced age, the degree of dilatation usually is insufficient as to be a cause of aortic valvular incompetence.

Rarely, we have observed established cases in the advanced age group in which roentgenograms showing pronounced dilatation of the ascending aorta and left ventricular enlargement in an elderly patient with clinical signs of aortic insufficiency may only be suspected as resulting from senile dilatation after excluding other conditions (Fig. 7).

B. TEARING OF THE ASCENDING AORTA

The ascending aorta is subject to tearing under several circumstances. The most common of these is typical dissecting aneurysm (Fig. 8*a*). The usual background for dissecting aneurysm is systemic hypertension.[2,13] Although cystic medial necrosis of the aorta is often considered a common association of dissecting aneurysm,[15] this process is frequently of minor degree.

After the initial tear occurs in the ascending aorta, a plane of cleavage is developed within the aortic media by a dissecting hematoma. The tear in the ascending aorta, if located just above the aortic valve, may result in aortic incompetence by virtue of loss of support of one or more of the aortic cusps (Fig. 8*b*).

In some instances the internal tear, like that of dissecting aneurysm, is not followed by a dissecting hematoma (Fig. 8, *a* and *b*). The aortic wall, however, is weakened and a saccular aneurysm may develop. (This type of aneurysm is referred to as an "incomplete dissecting" type of aneurysm.)

In either instance, when valvular incompetence complicates a tear of the ascending aorta, the functional abnormality results either from dilatation of the aorta and valvular distortion (as in syphilitic aortitis), or the tear is so located as to cause loss of support of one or more of the aortic cusps.

The roentgenologic findings to be dis-

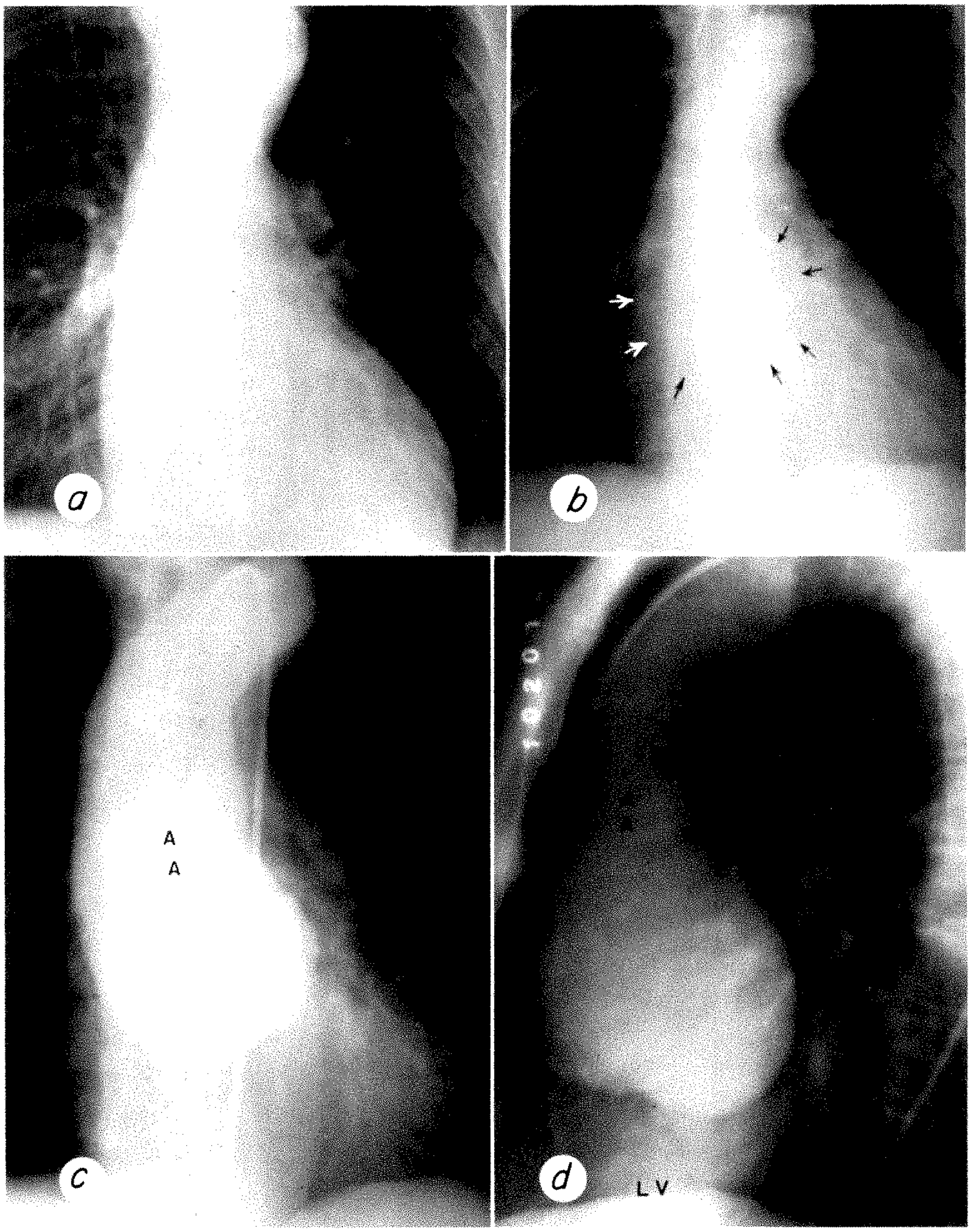

Fig. 6. Idiopathic dilatation of the ascending aorta with aortic valvular regurgitation in a 37 year old man. Abbreviations as in Figure 2. (*a*) Frontal thoracic roentgenogram showing enlarged left ventricle and suggestion of only a mild degree of dilatation of the ascending aorta. (*b*) Heavily penetrated frontal roentgenogram shows a mass density (arrows) representing the ascending aorta overlying the mid-dorsal spine. (*c* and *d*) Frontal and lateral thoracic aortograms showing marked dilatation of the aortic root including the aortic valvular ring and aortic sinuses. There is severe aortic valvular regurgitation. The dilated aortic sinuses and lower ascending aorta correspond to the "mass lesion" shown in *b*.

cussed will apply only to that small group of patients who survive the initial episode of a tear in the aorta and then develop aortic valvular insufficiency.

1. *Complete Dissecting Aneurysm.* When valvular incompetence complicates a dissecting aneurysm, the initial tear usually occurs just above the aortic valve rather than at distal levels.

The roentgenologic appearances are de-

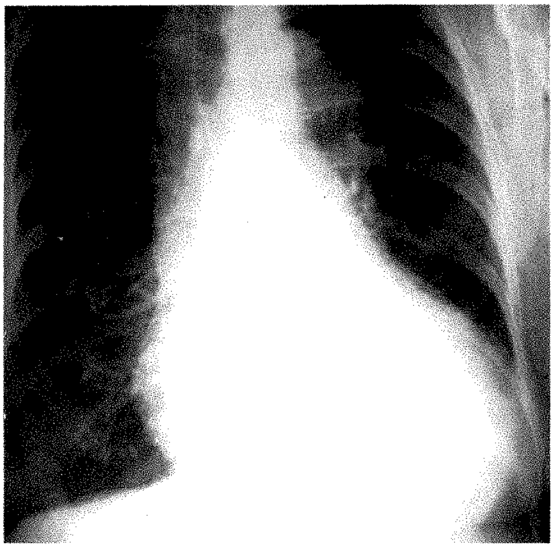

Fig. 7. Senile dilatation of the ascending aorta with aortic valvular regurgitation in a 68 year old man. Thoracic roentgenogram shows prominence of the ascending aorta and of the aortic arch and left ventricular enlargement.

termined by the stage at which the examination is made. In all stages, the ascending aorta is dilated and the aortic knob is abnormally high and large (Fig. 9). The heart is enlarged owing to the aortic valvular incompetence that has developed. In the lateral view, the ascending aorta bulges superiorly into the retrosternal space. If previous roentgenograms are available that show a linear plaque in the wall of the ascending aorta or of the aortic knob, the plaque may serve as a baseline from which to measure a change in the diameter of the aorta. The roentgenographic appearance may be difficult to distinguish from syphilitic aortitis when calcification has developed along the length of the aneurysm, as it may in cases of "healed" dissecting aneurysm.

Dissecting aneurysm can best be delineated by aortography. Its appearance is characterized by a ragged, narrowed aortic lumen accompanied by a thickened aortic wall. In the presence of such changes, associated aortic regurgitation lends further support to the diagnosis of dissecting aneurysm.

2. *Incomplete Dissecting* (*Saccular*) *Aneurysm.* A localized saccular aneurysm resulting from a tear in the aorta rarely occurs in

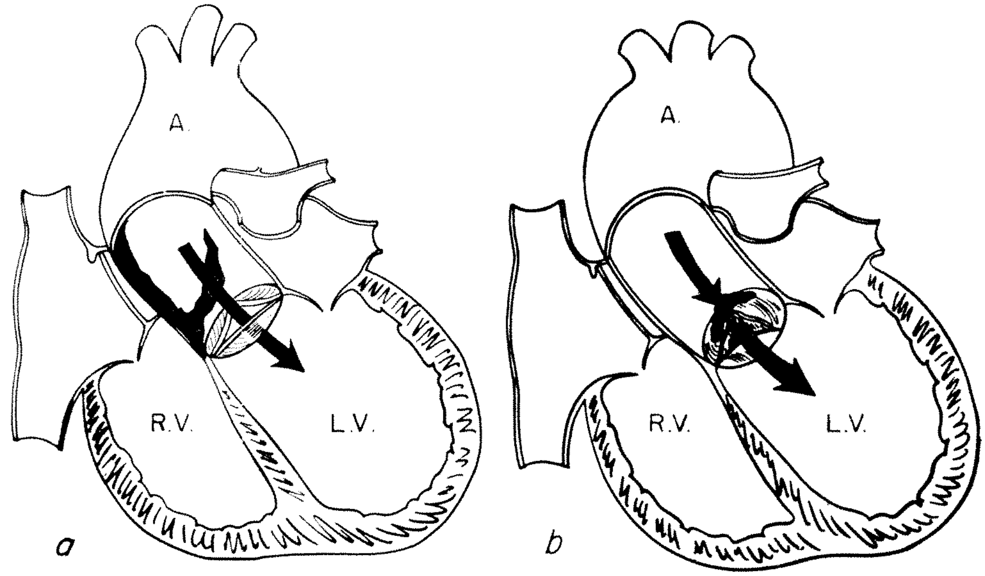

Fig. 8. Schematic representation of the two fundamental types of tear which occur in the ascending aorta. The shaded area represents blood in the media of the aortic wall. Large arrows indicate aortic insufficiency. (*a*) Dissecting aneurysm. Dilatation of the aorta causes aortic regurgitation. (*b*) Incomplete dissecting (saccular) aneurysm. Tear above ascending aorta has not resulted in dissection. Support of aortic valve is weakened.

the ascending portion of the vessel. The closer the tear is to the aortic valve, the more likely will be the chances that aortic regurgitation will appear.

A saccular aneurysm of the ascending aorta either may appear as a localized prominence of the ascending aorta or cause no alteration in configuration of the ascending aorta (Fig. 10, *a* and *b*). The absence of roentgenographic abnormality may be indicative of origin of the aneurysm from the posterior aspect of the aortic wall. Calcification may develop in the wall of the aneurysm causing it to be confused with syphilitic aortitis.

Selective aortography is of extreme aid in showing both the localized saccular dilatation above the aortic sinuses and the degree of aortic incompetence. Without aortography, the differentiation between saccular aneurysm and aneurysmal dilatation of the ascending aorta on the basis of syphilis, cystic medial necrosis or intrinsic disease of the aortic valve is difficult.

The case shown in Figure 10, *a* and *b* was studied at necropsy and reported by Levy and associates.[19] This was a 49 year old man with an "incomplete dissecting" aneurysm of the ascending aorta, the result of cystic medial necrosis. As the diseased part of the aorta became dilated, it stretched the attached aortic cusps. As a result, one cusp developed a gross fenestration, while a second ruptured and prolapsed.

II. RUN-OFF OF BLOOD THROUGH CHANNELS OTHER THAN AORTIC VALVE

The first section of this communication dealt with conditions of the ascending aorta which caused incompetence of the aortic valve while the aortic valve was, as a rule, intrinsically normal. The conditions forming the basis for this section have, in common with the above noted diseases, an abnormal run-off of blood from the ascending aorta which may simulate functionally intrinsic aortic valvular disease. In contrast, however, in the conditions herein to be considered, the run-off of blood is not through the aortic valve but rather through an abnormal communication between the ascending aorta on the one hand, and a cardiac chamber or a large mediastinal vessel, on the other.

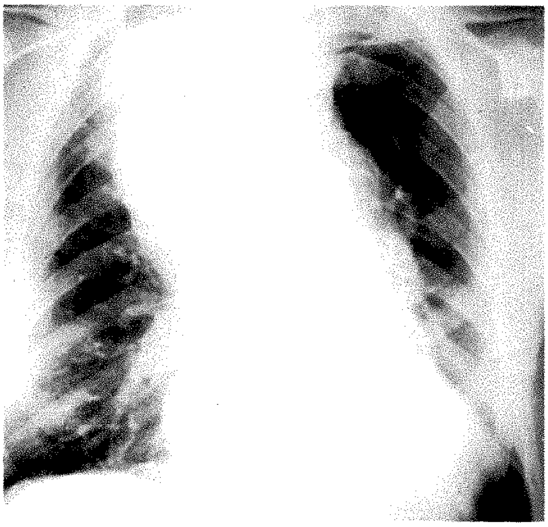

Fig. 9. Dissecting aneurysm of the ascending aorta causing aortic valvular regurgitation in a 64 year old man. Thoracic roentgenogram shows widening of the entire ascending aorta which obscures the medial portion of the right upper pulmonary field. The descending thoracic aorta shows an irregular and shaggy contour. In addition, there is left ventricular enlargement.

Abnormal communications involving the ascending aorta may arise either from the beginning of the aorta, at the sinuses or at more distal levels in this segment of the vessel. Congenital and inflammatory lesions are the most common causes.

ANEURYSMS OF THE AORTIC SINUSES

Aortic sinus aneurysms are of two types, the congenital and the acquired.[25] The pathologic changes in congenital aortic sinus aneurysm consist primarily in separation of the aortic media from the annulus fibrosus of the aortic valve.[7] This separation causes a weakness of the wall of the aorta and aneurysmal protrusion follows. Following this development, rupture of the aneurysm may occur.

Acquired aneurysms of the aortic sinuses usually are of mycotic nature secondary to bacterial endocarditis, particularly that

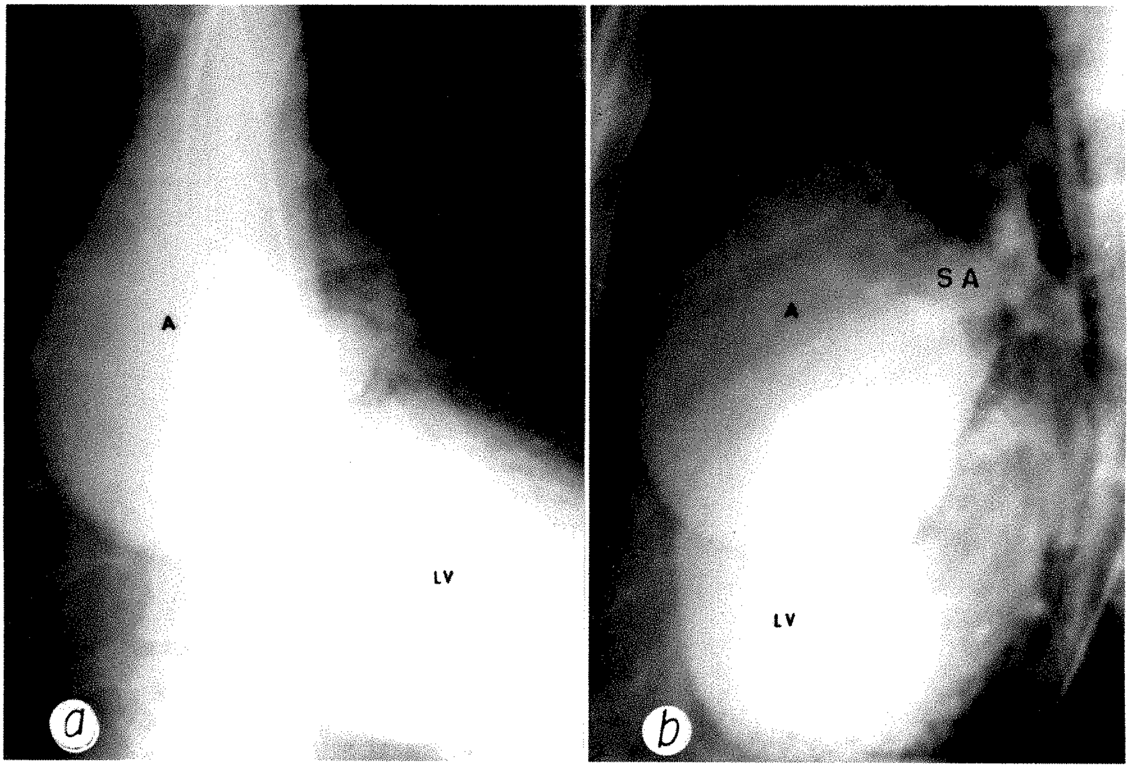

FIG. 10. Thoracic roentgenograms and selective thoracic aortogram in a 49 year old man with aortic insufficiency secondary to saccular aneurysm of ascending aorta of the "incomplete dissecting" aneurysm type. Aneurysm was result of cystic medial necrosis. Case reported by Levy and associates.[19] (*a*) Frontal and (*b*) lateral views, respectively. There is marked dilatation of the lower portion of the ascending aorta (A). In contrast to the uniform dilatation of the ascending aorta seen in cystic medial necrosis without saccular aneurysm, there is an eccentric saccular projection (SA) of the left and posterior aspects of the ascending aorta just above the left aortic sinus. Severe aortic insufficiency is shown by dense opacification of the left ventricle (LV). (From Levy and associates[19] with permission.)

involving the aortic valve. When mycotic aneurysms rupture, they do so into the structure or chamber which happens to be contiguous with the aneurysm. In contrast to the usual congenital aneurysm, mycotic aneurysms may, in a rare instance, rupture into the pericardial space.

The most common location for aortic sinus aneurysm is the right aortic sinus.[7,16,25] Commonly, it ruptures into the outflow tract of the right ventricle, and frequently is associated with a ventricular septal defect (Fig. 11*a*). Less commonly, an aneurysm of the right sinus ruptures into the right atrium. Next in order of frequency are aneurysms of the posterior aortic sinus (Fig. 11*b*). These usually present or rupture into the right atrium and commonly occur as isolated malformations.

Aneurysms involving the left aortic sinus are extremely rare. By virtue of its anatomic location, the aneurysm at this site may cause narrowing of the left coronary artery,[8] or it may rupture into the pericardial cavity or into the left atrial appendage.[14]

When aneurysms of an aortic sinus rupture, they exhibit abnormal run-off of blood during systole and diastole, and, in some aspects, may be confused with aortic insufficiency.

It should be emphasized that the right and posterior aortic sinuses, by virtue of their location, are not border-forming structures in the cardiovascular silhouette, even following aneurysmal dilatation. Rarely, however, an aneurysm of the left aortic sinus will appear as a small bulge in

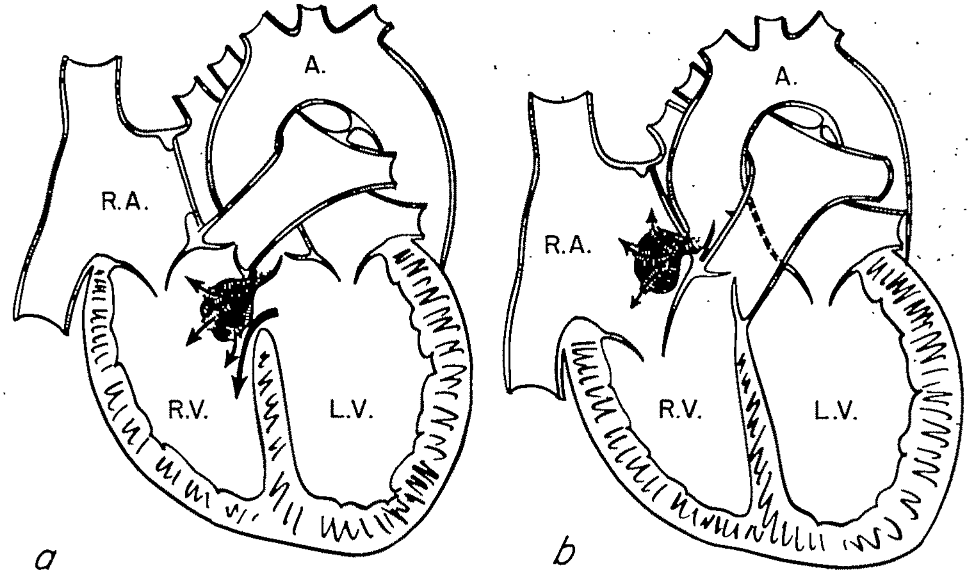

Fig. 11. Diagrammatic representation of the two common forms of congenitally ruptured aortic sinus aneurysm. (Modified from Kanjuh and Edwards[17] with permission.) (*a*) Aneurysm of right aortic sinus. ruptured into the right ventricle (R.V.). Commonly, a ventricular septal defect (bold arrow) is associated. (*b*) Aneurysm of posterior aortic sinus ruptured into the right atrium (R.A.).

the upper portion of the left cardiovascular silhouette resembling a prominent left atrial appendage.[8]

The roentgenologic findings depend upon the size of the communication between the ascending aorta and the cardiac chamber with which it communicates. Therefore, the roentgenographic findings may range from normal in the case of a small communication to those of a large left-to-right shunt with considerable pulsation of the entire thoracic aorta when the communication is large.

The roentgenologic features among the few cases observed by us were identical with those of a large left-to-right shunt occurring distally to the atrioventricular valves, regardless of whether the rupture occurred into the right atrium or into the right ventricle (Fig. 12, *a*, *b* and *c*).

In the case of aneurysms of the right aortic sinus, these findings are not unexpected, since this type is usually associated with a ventricular septal defect (Fig. 12, *a*, *b* and *c*). Moreover, roentgenoscopy did not reveal any abnormal pulsations of the thoracic aorta or right atrium.

Thoracic aortography is the only definitive means of diagnosis. As shown in Figure 12*c*, the contrast-filled right aortic sinus aneurysm may be seen to herniate into the outflow tract of the right ventricle. A small jet of opacified blood enters the right ventricle through the defect in relation to the right aortic cusp.

AORTICO-LEFT VENTRICULAR TUNNEL

This condition may be congenital or acquired in origin. Both types are to be distinguished from aortic sinus aneurysm. The congenital form is represented by an abnormal vessel-like structure which begins in the anterior aspect of the ascending aorta (above the right coronary artery), bypasses the aortic valve, and leads through the ventricular septum into the left ventricular cavity (Fig. 13). Within the ventricular septum, the tunnel lies in the posterior wall of the right ventricular infundibulum. In 1 of 3 cases reported by Levy and associates,[18] the extracardiac portion of the tunnel exhibited a saccular aneurysm. These authors observed that histologic examination of the ascending aorta showed none of

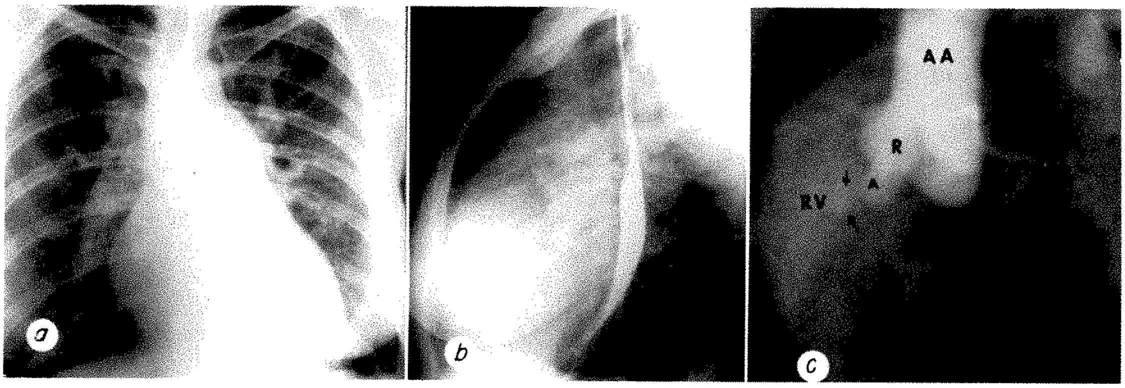

Fig. 12. Ruptured right aortic sinus aneurysm communicating with the right ventricle in association with a ventricular septal defect. (*a* and *b*) Frontal and lateral thoracic roentgenograms. The pulmonary trunk and central and peripheral pulmonary arteries are prominent, indicating a left-to-right shunt. Left atrial enlargement indicates that the shunt is distal to the atrioventricular valves. Scoliosis of upper dorsal spine results in the aortic arch being somewhat prominent. (*c*) Selective thoracic aortogram in lateral view showing the aneurysm (A) of the right aortic sinus (R) projecting into the right ventricle (RV). Contrast material (arrows) is shown passing from the aneurysm into the right ventricle. AA = ascending aorta.

the features encountered in Marfan's syndrome.

The roentgenograms in each of the 3 cases of congenital aortico-left ventricular tunnel reported by Levy and associates[18] showed moderate to severe cardiomegaly in the frontal projection. Enlargement was principally of the left ventricle as evidenced by a prominent left lower cardiac border. There was also a small convex bulge in the region of the shadow formed by the pulmonary trunk. There was absent to mild left atrial enlargement. The pulmonary vasculature was normal. The most significant finding was pronounced dilatation of the ascending aorta. Roentgenoscopically, the entire thoracic aorta revealed prominent pulsations, identical with those seen in aortic valvular incompetence.

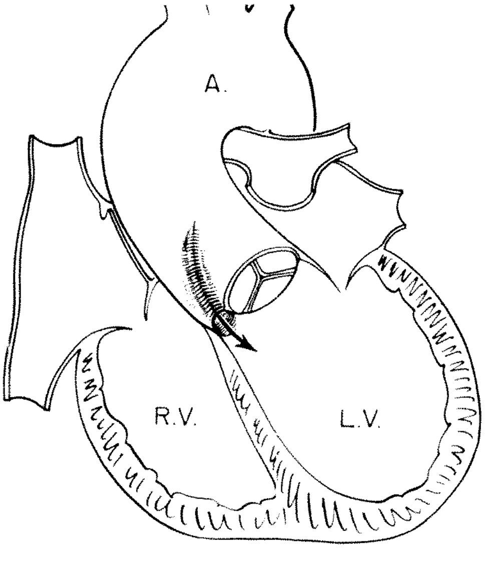

Fig. 13. Aortico-left ventricular tunnel. Schematic representation showing the vessel-like structure between the ascending aorta and the left ventricle.

Specific diagnosis is derived from an ascending thoracic aortogram. The ascending aorta shows fusiform dilatation. In the aortogram, visualization of a vessel-like structure above the right aortic sinus and projection into the region of the pulmonary trunk is the typical finding. Lack of contrast material entering the pulmonary arteries together with opacification of the left ventricle should establish the correct diagnosis. On occasion, aortic valvular regurgitation coexists.

Infection of the aorta usually is a complication of bacterial infection of the aortic valve (Fig. 14). Weakening of the aortic wall by infection may result in a mycotic aneurysm. Rupture of the aneurysm leads to abnormal escape of blood from the ascending aorta.[26] Because of the potential complexity of the path that an infectious process may take, exotic channels may result from bacterial infection. Communi-

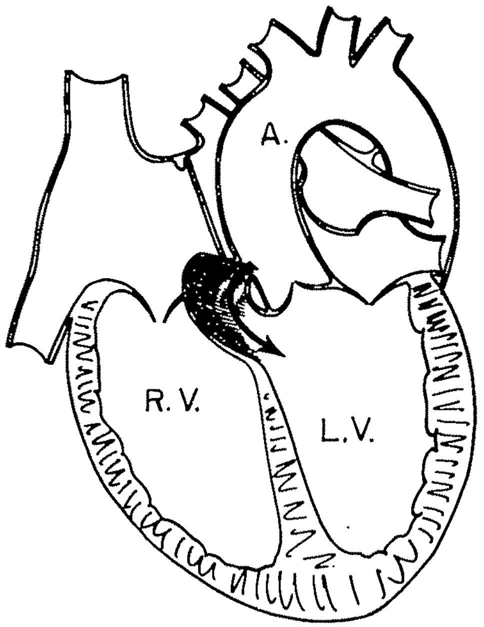

Fig. 14. Acquired form of aortico-left ventricular communication. Schematic representation of mycotic aneurysm of aorta communicating with the left ventricle as a complication of bacterial endocarditis.

cation may be established between any adjacent structure, such as either ventricle or atrium, the pulmonary arterial system, the superior vena cava or the pericardium.

The roentgenologic findings are not characteristic, since the eventual structural effects depend on the site into which the aneurysm ruptures. The diagnosis may be suspected when a patient with a history of bacterial endocarditis suddenly develops signs of aortic insufficiency or a left-to-right shunt.

Specific diagnosis of an aneurysm with abnormal run-off of blood into the left ventricle is derived from thoracic aortography. The unruptured aneurysm resulting from bacterial infection may be difficult to distinguish from an incomplete dissecting (saccular) aneurysm.

SYPHILITIC AORTITIS

Although syphilitic aortitis classically causes incompetence of the aortic valve, an additional complication is rupture of the aorta or of an aneurysm into adjacent structures, such as the superior vena cava (Fig. 15*a*), a pulmonary artery (Fig. 15*b*), or the pericardial cavity. Rupture into a vessel allows an abnormal run-off from the aorta at a level distal to the aortic sinuses.

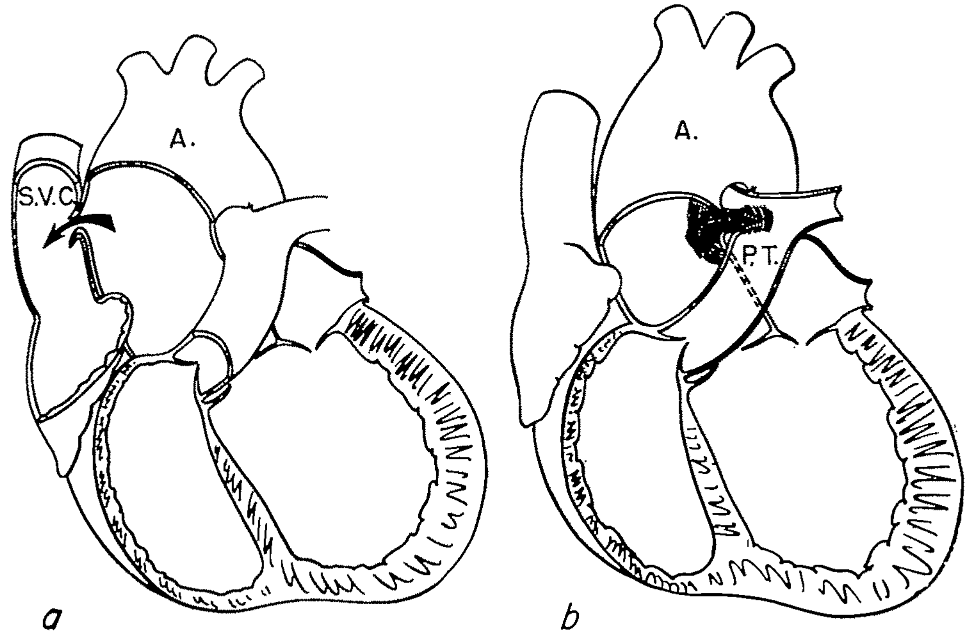

Fig. 15. Schematic representation of syphilitic aneurysm rupturing into (*a*) the superior vena cava (S.V.C.) and (*b*) the pulmonary trunk (P.T.).

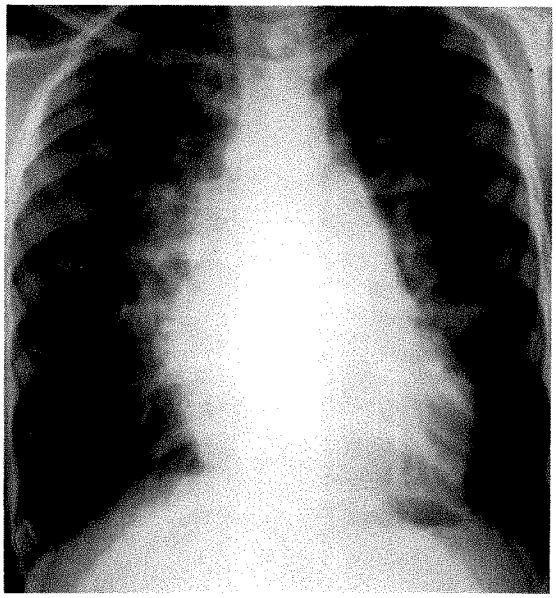

FIG. 16. Aorticopulmonary septal defect. Thoracic roentgenogram in the frontal view showing prominent pulmonary trunk, central and peripheral pulmonary vessels, indicating a left-to-right shunt.

The proper diagnosis might be suspected from study of roentgenograms if the subject has exhibited the classic signs of syphilitic aortitis prior to rupture of the aneurysm. The fundamental complicating signs are those of a left-to-right shunt and right-sided cardiac enlargement. Rupture into the superior vena cava would result in prominence of this structure. In the thoracic aortogram, visualization of the aneurysm and its site of communication should, in most cases, establish the diagnosis of syphilitic aneurysm communicating with a contiguous vessel. It is conceivable that serologic tests for syphilis might be necessary to differentiate this condition from mycotic aneurysm, since in each of the conditions the wall of the aneurysm may calcify.

AORTICOPULMONARY SEPTAL DEFECT

This congenital lesion is characterized by a window-like communication between the ascending aorta and pulmonary arterial trunk. The opening lies in the left anterior wall of the aorta just above the level of the left coronary arterial ostium. In the pulmonary trunk, the opening is located in its right wall just anterior to the ostium of the right pulmonary artery. In general, the hemodynamic findings are similar to those in cases of large patent ductus arteriosus. The latter condition, in fact, is associated with about 12 per cent of cases of aorticopulmonary septal defect.[22]

The roentgenographic appearances are those of a left-to-right shunt, namely, dilatation of the pulmonary trunk and its branches. These vessels may exhibit conspicuous pulsations during roentgenoscopy (Fig. 16). The heart is moderately enlarged and the left atrium is slightly enlarged. Roentgenograms indicate a left-to-right shunt distal to the atrioventricular valves. In contradistinction to patent ductus arteriosus, there usually is no prominence of the aortic knob.

Roentgenologically, a specific diagnosis may only be derived by selective thoracic aortography.[12,22] By this technique, the ascending aorta and the pulmonary trunk are noted to be connected by a window-like defect (Fig. 17, *a* and *b*) and a separation between the two vessels below the defect will usually be seen. It is essential to identify both the aortic and pulmonary valves since in persistent truncus arteriosus the findings may be similar except for the fact that there is only one semilunar valve in the latter condition.

CORONARY FISTULA

The communication of a coronary artery with a cardiac chamber, coronary sinus, or the pulmonary trunk may cause functional derangements that allow an abnormal run-off of blood from the ascending aorta during systole and diastole (Fig. 18, *a–d*).[21] Only if the anomalous coronary artery connects with the left ventricle does the run-off occur only during diastole. These anomalies may be complicated by bacterial infection, myocardial ischemia, congestive cardiac failure, or all three. The right coronary artery is involved slightly more commonly than the left.[11,21] The sites of termination are most often the right atrium or a

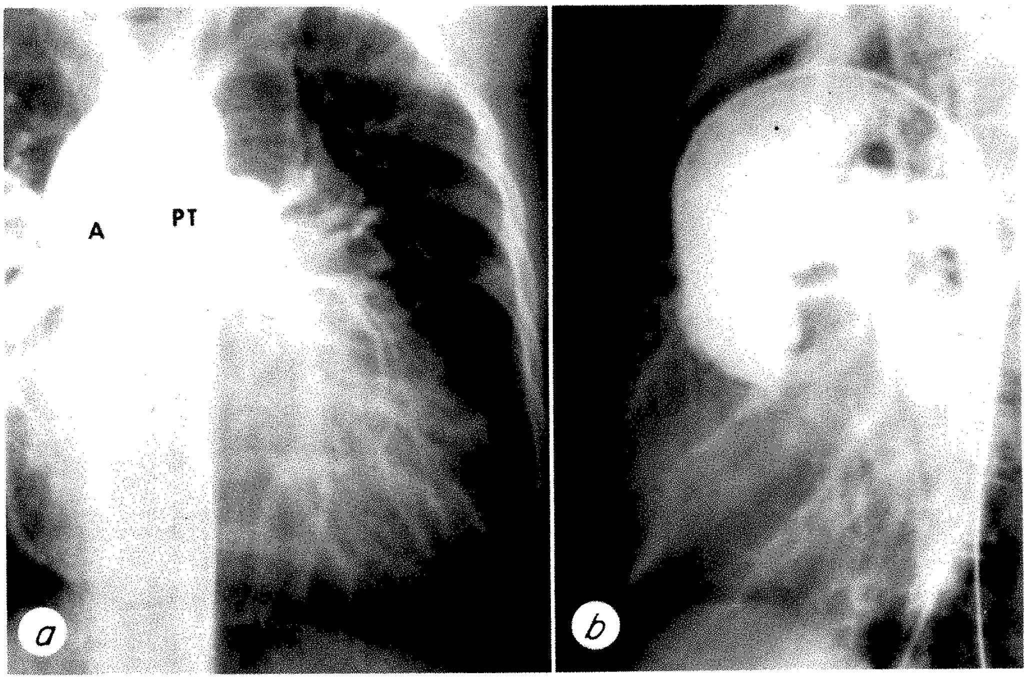

Fig. 17. Selective thoracic aortograms in aorticopulmonary septal defect. (*a*) Frontal and (*b*) lateral views. There is opacification of the ascending aorta (A) and a shunt of contrast material from the ascending aorta through the aorticopulmonary septal defect into the pulmonary trunk (PT).

cardiac vein, the right ventricle and the pulmonary trunk (Fig. 18, *a–d*).

The roentgenograms commonly reveal evidence of a left-to-right shunt (Fig. 19, *a* and *b*), but if the shunt is small the roentgenograms may yield normal features. If myocardial ischemia is a complicating feature, the roentgenograms show signs consistent with a cardiac myopathy rather than a left-to-right shunt.

When a left-to-right shunt is evident, the left atrium is enlarged in approximately one-half of the cases.[21] The aortic knob is not conspicuously prominent. In general, the roentgenologic findings are not diagnostic and seem to depend on the hemodynamic alterations in the individual case.

On rare occasions, peculiar densities or a prominence along the border of the cardiac silhouette may be caused by the involved dilated and tortuous coronary artery. In a case reported by Barcia and associates,[1] the enormous dilatation of the right coronary artery was represented by an unusual and tortuous density within the right aspect of the cardiac silhouette. In a case reported by Neufeld and associates,[21] the enlarged left coronary artery yielded an abnormal prominence of the mid-left cardiac border.

A specific diagnosis derived by thoracic aortography characteristically shows that the coronary artery which enters into anomalous communication with a cardiac chamber or pulmonary trunk is obviously enlarged. It is not only dilated, but elongated and tortuous. The enlarged artery commonly shows focal saccular aneurysms with calcified walls. Filling of the anomalous coronary vessels (the right or left) is followed by opacification of the chamber or vessel with which the artery is in anomalous communication.

ANOMALOUS ORIGIN OF THE LEFT CORONARY ARTERY FROM THE PULMONARY TRUNK

In this condition, the right coronary artery arises from the aorta in its usual position, whereas the left coronary artery arises from the pulmonary trunk. Endocardial sclerosis of the left ventricle is often present. Coexisting mitral insufficiency on the basis of infarction of the papillary

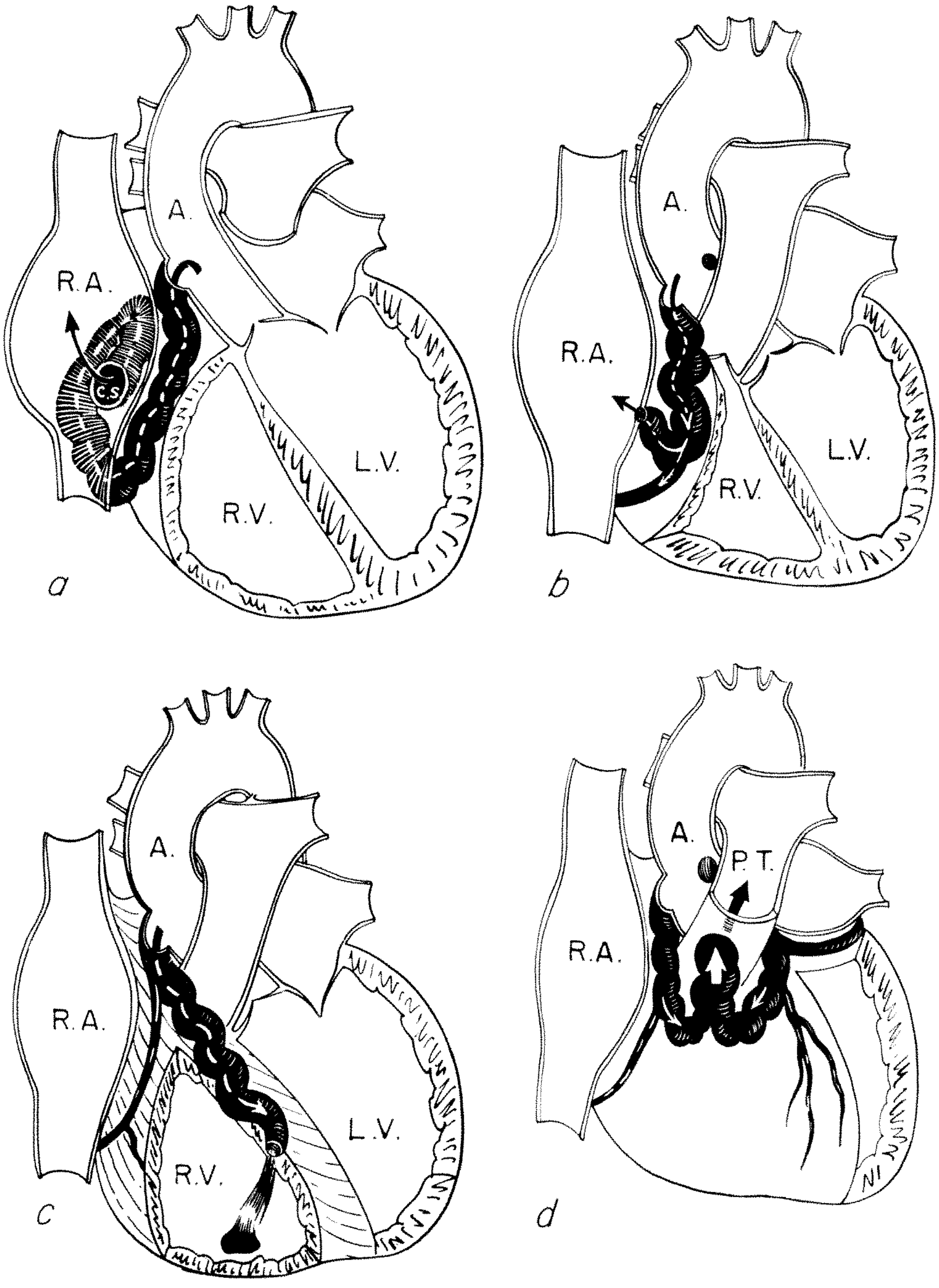

Fig. 18. Anomalous connections of coronary arteries. Schematic representation of conditions in which two coronary arteries arise from the aorta and in which one or both vessels make anomalous connection with a cardiac chamber or a vessel. (Modified from Kanjuh and Edwards[17] with permission.) (*a*) Right coronary artery joins coronary sinus. (*b*) Right coronary artery connects with right atrium (R.A.). (*c*) Right ventricle (R.V.) receives a branch of the right coronary artery. (*d*) Both coronary arteries connect through an "accessory coronary artery" with the pulmonary trunk (P.T.).

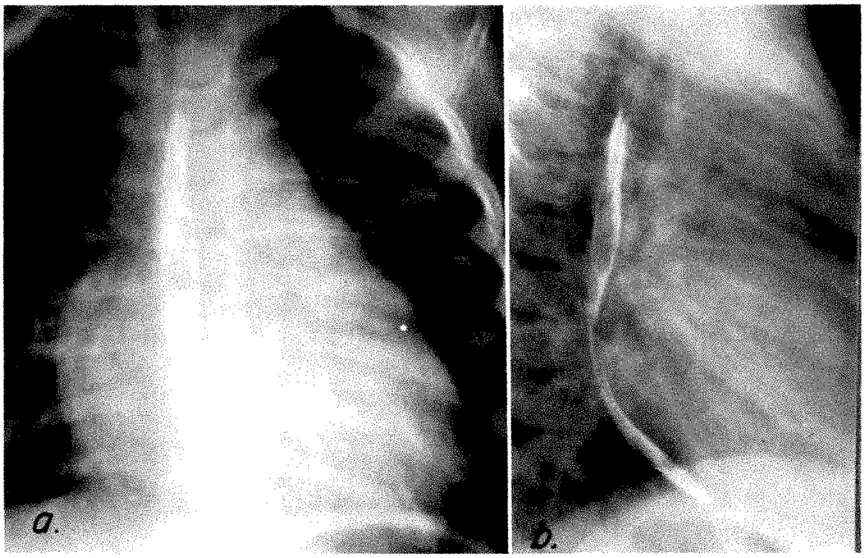

Fig. 19. Coronary arterial fistula to right ventricle. (*a*) Frontal and (*b*) lateral roentgenograms. The roentgenographic findings are simply those of a left-to-right shunt distal to the atrioventricular valves.

muscles of the mitral valve may occur.[23]

Anomalous origin of the left coronary artery in the older infant and child is similar functionally to the usual coronary fistula. In both of these situations, an arteriovenous-like arrangement involves the coronary arterial system and is reflected as an abnormal escape of blood from the aorta to the pulmonary trunk. The direction of blood flow is governed by the fact that the resistance to the flow of blood in the pulmonary circulation is less than that in the systemic circulation.

The roentgenogram shows marked cardiomegaly owing to left ventricular enlargement (Fig. 20). The left atrium is usually mildly enlarged and the pulmonary vasculature is prominent, indicating pulmonary venous congestion. These findings are identical to those observed in endocardial sclerosis and mitral insufficiency.

The most definitive diagnostic procedure is selective right coronary arteriography. Injection into the right coronary artery ultimately shows opacification of the pulmonary trunk. In the first sequence of films, the right coronary artery is large and tortuous. This is followed in rapid sequence by filling of anastomotic twigs, then reversed flow through the left coronary artery into the pulmonary trunk.

COMMENT

In this communication, the roentgenographic findings in diseases of the ascending aorta responsible for abnormal run-off of blood were discussed.

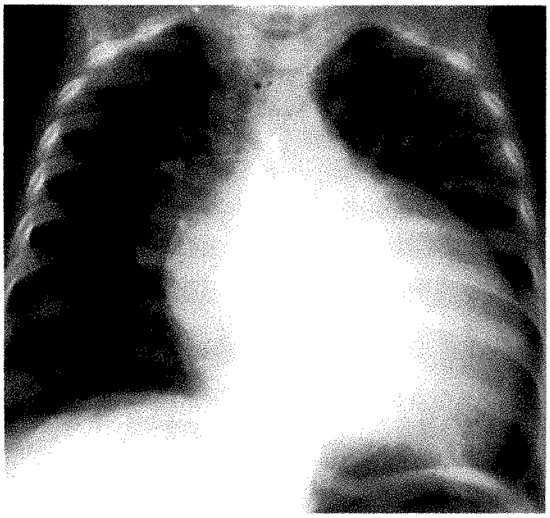

Fig. 20. Anomalous origin of the left coronary artery. Frontal thoracic roentgenogram showing cardiomegaly principally by virtue of left ventricular and left atrial enlargement.

In the first major category of diseases of the ascending aorta, abnormal dilatation of the ascending aorta resulting from intrinsic weakness or from a tear causes mechanical distortion of the aortic valve and the latter may close improperly. It was indicated that the clinical and roentgenologic pictures might be confused with those of incompetence of the aortic valve owing to intrinsic aortic valvular disease. These roentgenographic features are (a) dilatation of the ascending aorta, (b) left ventricular enlargement and (c) increased pulsations of the entire thoracic aorta.

Although certain functional and structural effects of these two anatomic forms of aortic insufficiency are alike, certain other findings in the roentgenogram often serve to distinguish one disease process from another. Pronounced dilatation of the ascending aorta usually indicates a primary condition of the ascending aorta, whereas aortic valvular regurgitation owing to intrinsic valvular disease usually results in only mild to moderate dilatation of the ascending aorta. Additional signs which support a diagnosis of intrinsic disease of the aortic valve (whereas absence of these signs indicates a condition of the ascending aorta) are (d) coexisting mitral valvular disease, (e) calcification in the aortic valve and (f) signs of coexisting aortic valvular stenosis.

When a condition involving the ascending aorta primarily is suspected, certain other roentgenologic findings, when present, strongly suggest the specific disease process involving the ascending aorta. These are as follows:

(1) *Syphilitic aortitis* may often be diagnosed from roentgenograms on the basis of the commonly associated intimal calcification of the ascending aorta, of the aortic sinuses, or of both.

(2) Aortic insufficiency on the basis of *rheumatoid spondylitis* should be suggested if the specific skeletal changes are present.

(3) Aortic insufficiency on the basis of *cystic medial necrosis* of the aorta may be considered with confidence when the physical and skeletal changes of arachnodactyly, subluxed lens, etc., are present. In the so-called *forme fruste* of Marfan's syndrome, termed idiopathic dilatation of the ascending aorta, the roentgenologic findings are identical to those in arachnodactyly except that the associated skeletal and physical findings are absent. In spite of the presence of aortic dilatation, roentgenograms in the frontal view may fail to show this feature. In such cases the dilatation of the ascending aorta is usually apparent in lateral or left anterior oblique views. Frequently, the frontal projection shows a mass-density over the dorsal spine.

The rare heritable disorders resulting in dilatation of the ascending aorta may be diagnosed only if the typical skeletal and clinical findings coexist.

(4) When aortic valvular incompetence complicates a tear in the ascending aorta, the predominant roentgenologic signs may be those of *dissecting aneurysm*. When the tear results in a so-called "incomplete dissecting" aneurysm, diagnosis may be difficult from the thoracic roentgenogram.

In the second major group of conditions discussed herein, namely, those characterized by an abnormal communication between the ascending aorta, on one hand, and a cardiac chamber or a large mediastinal vessel, on the other, the functional effects are roentgenographically more variable than those disease processes of the ascending aorta causing abnormal dilatation. In some, the roentgenographic findings indicate a left-to-right shunt; in others, there appears to be aortic valvular regurgitation. The conditions in the second group which simulate aortic valvular regurgitation are represented by congenital and acquired forms of communication between the aorta and left ventricle.

The roentgenographic signs in the majority of conditions allowing run-off of blood through channels other than the aortic valve are nonspecific as to whether the primary disease process resides in the ascending aorta. On the other hand, the auscultatory findings at times yield considerable information as to the functional nature of the disease process. When these are correlated with the roentgenographic findings, they may be significant in indicating that the fundamental site of disease lies in the ascending aorta.

Shunts in which the site of origin is at the aortic level characteristically yield a continuous or near-continuous murmur. The reason for this is apparent when one recalls that this hemodynamic situation allows near-continuous run-off of blood throughout the cardiac cycle from the aorta to a vascular compartment having venous pressure.

When a patient with roentgenographic signs of a left-to-right shunt exhibits a continuous or near-continuous murmur, the radiologist should be alerted to the fact that the fundamental site of disease may lie in the ascending aorta. Performance of selective thoracic aortography is the ideal method of establishing an exact diagnosis.

SUMMARY

Conditions of the ascending aorta responsible for abnormal run-off of blood when viewed roentgenographically may be divided into 2 major categories: (I) those allowing a run-off of blood through the aortic valve and (II) those characterized by an abnormal communication between the ascending aorta, on one hand, and a cardiac chamber or a mediastinal vessel, on the other. In the former situation, the roentgenograms usually indicate that the region in or around the ascending aorta is the site of disease and the decision to perform selective aortography is obvious. In the second major group, the fact that the site of the disease process begins primarily in the ascending aorta is not obvious from conventional roentgenographic data. Correlation, however, of the roentgenographic evidence for a left-to-right shunt with the auscultatory findings of a systolic and diastolic murmur indicates that selective thoracic aortography is of fundamental importance in establishing the correct diagnosis.

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KINKING OR BUCKLING OF THE AORTA*

CASE REPORT WITH COMPLICATION OF ANEURYSM FORMATION

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KINKING or buckling of the aorta is an uncommon anomaly in which the descending portion of the aortic arch at the level of the ligamentum arteriosum is acutely kinked. The aorta proximal to the kink is elongated and arises higher in the mediastinum than usual. This anomaly is asymptomatic and has previously been considered to be a benign condition, although it often causes difficulty in the interpretation of the chest roentgenogram as it presents as a left superior mediastinal mass.[3,4]

The purpose of the authors is to report a case followed for 12 years, during which time an aneurysm developed at the site of the kinking, which was subsequently successfully repaired.

REPORT OF A CASE

M.P., a 36 year old white female, was admitted to Los Angeles County General Hospital on December 10, 1953 for an incomplete abortion. On physical examination, her blood pressure was 130/85 and her radial and femoral pulses were normal. All laboratory data, including Wassermann anticomplementary and Kahn precipitation tests, were normal. On the admission chest roentgenogram (Fig. 1), a 3 cm. left superior mediastinal mass was noted just above the aortic arch. The patient left the hospital before further work-up could be obtained. She was re-admitted on March 16, 1958 for an incomplete abortion. On physical examination, her blood pressure was 140/80 mm. Hg in the right arm, 110/70 in the left arm, and 160/120 mm. Hg in both legs. A Grade 4/6 systolic murmur was heard best in the left infraclavicular region and transmitted to the apex. No thrills were palpable. A chest roentgenogram (Fig. 2) again demonstrated an irregular superior mediastinal mass which displaced the aortic arch slightly. At fluoroscopy, the density could not be separated from and appeared to pulsate with the aortic arch. A right brachial retrograde aortogram (Fig. 3) showed the typical appearance of a buckled aorta with the anterior arch elongated and elevated and with traction at the level of the ligamentum arteriosum indenting the posterior arch. There was no appreciable collateral flow via the intercostal, internal mammary, or periscapular arteries. The patient was discharged and followed in clinic with periodic chest roentgenograms. On January 21, 1965, the patient was re-admitted because of increasing size of the left superior mediastinal shadow (Fig. 4). Right axillary aortograms revealed a trilobulated aneurysm involving the aortic arch (Fig. 5, *A*

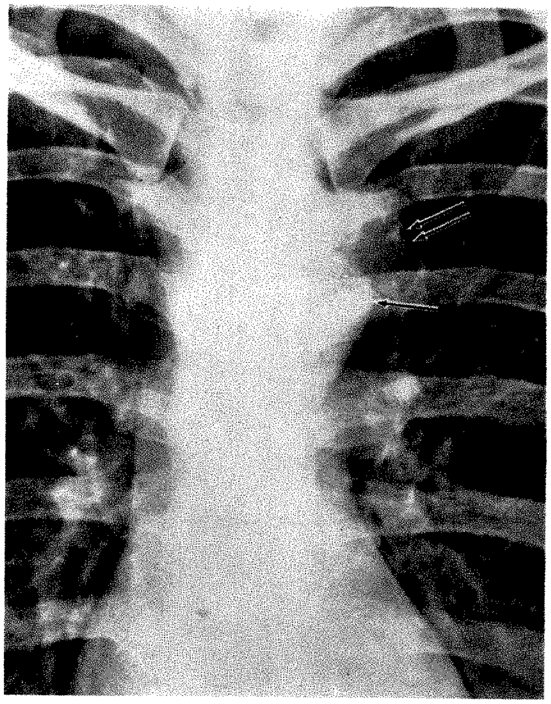

FIG. 1. *December 10, 1953.* There is elongation of the aortic arch (arrow) with small adjacent mass (double arrows).

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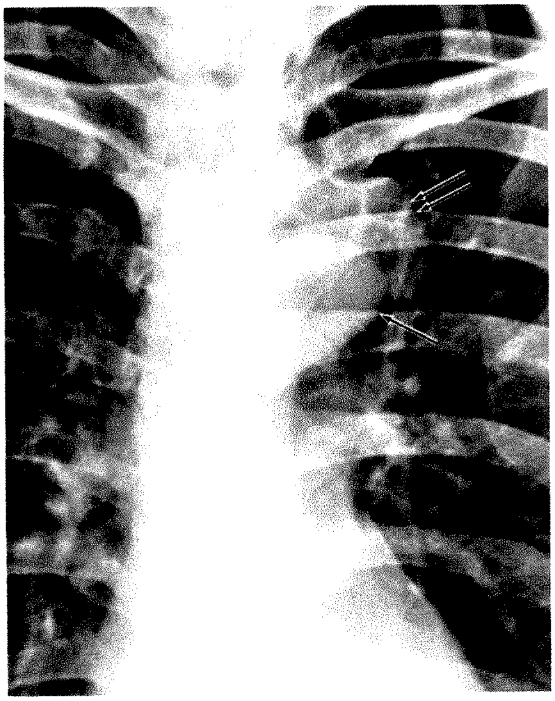

FIG. 2. *March 16, 1958.* Roentgenogram shows further elongation of the aortic arch (arrow) with no definite change in size of adjacent mass (double arrows).

and *B*). Again, there was no evidence of collateral circulation.

At surgery on Febuary 1, 1965, a typical pseudocoarctation was observed. A trilobulated aneurysm was found proximal to the ligamentum arteriosum (Fig. 6*A*). The walls of the aneurysm were so thin that the aortic blood flow could be observed through the aneurysm wall. The ligamentum arteriosum was doubly ligated and transected. The left subclavian artery was clamped and transected and the aorta was transected just distal to the left common carotid artery and distal to the aneurysm (Fig. 6*B*). The left subclavian artery was then reimplanted into the aorta (Fig. 6*C*). Microscopic sections of the aneurysm wall revealed no evidence of aortitis. The patient was dis-

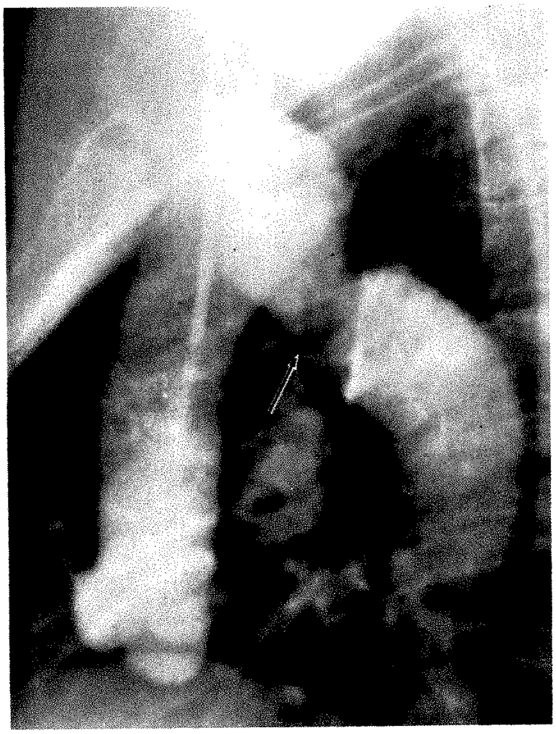

FIG. 3. *March 28, 1958.* Aortogram shows normal aortic valve and ascending aorta. There is folding of distal portion of aortic arch with kinking at level of ligamentum arteriosum (arrow). No poststenotic dilatation or collateral channels are noted.

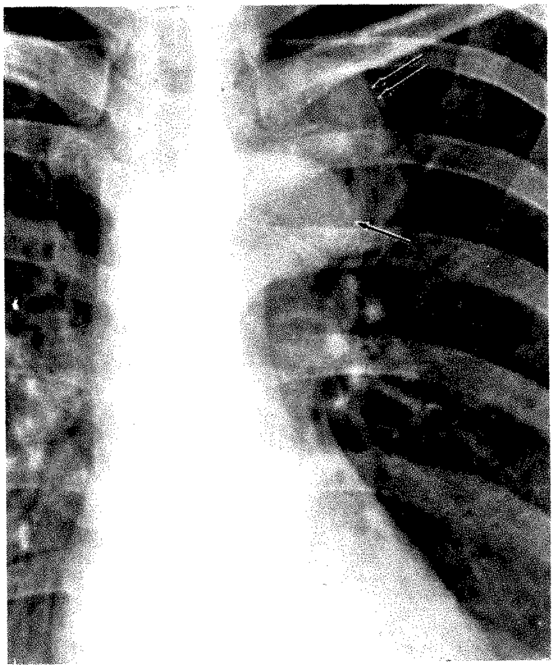

FIG. 4. *January 21, 1965.* There has been further elongation of the aortic arch (arrow) and the adjacent mass has shown a definite increase in size (double arrows).

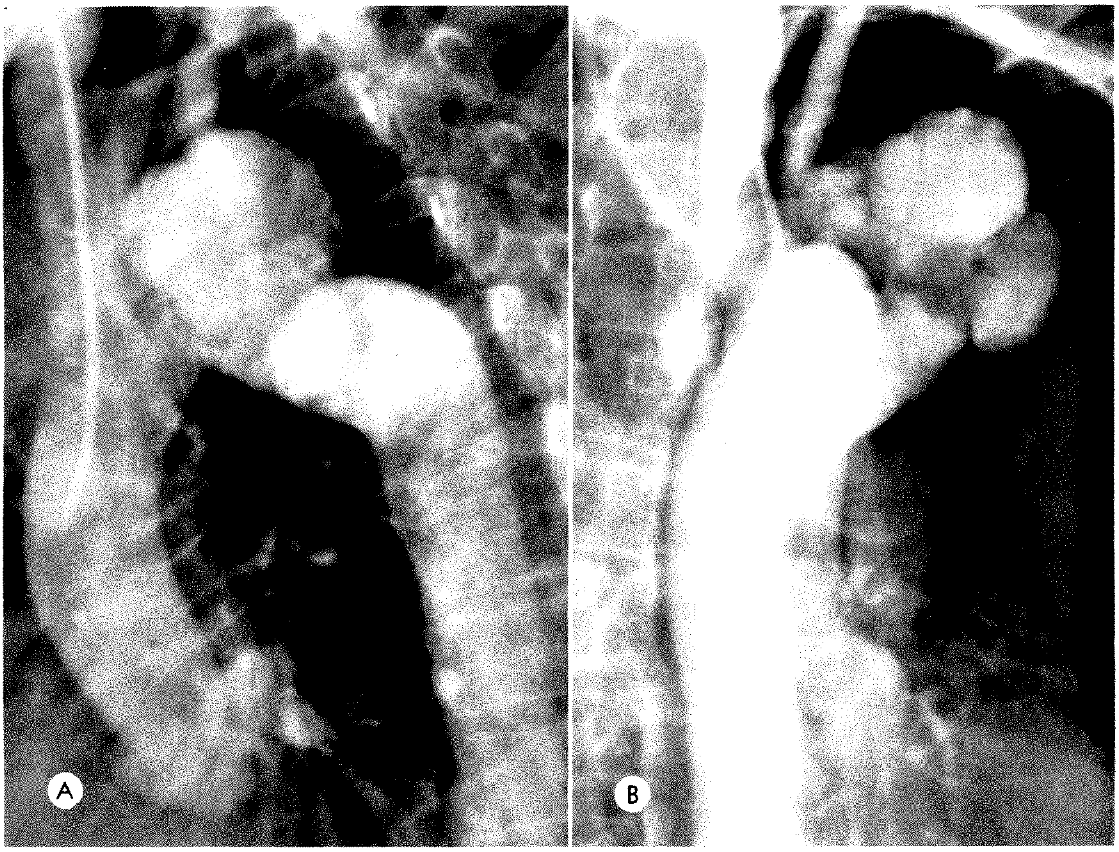

FIG. 5. *January 25, 1965.* (*A*) Percutaneous right axillary aortogram in left anterior oblique projection demonstrates lobulated aneurysm proximal to ligamentum arteriosum. (*B*) Right anterior oblique study more clearly demonstrates lobulated aneurysm.

charged on February 10, 1965 and is well at the present time.

DISCUSSION

Approximately 50 cases of pseudocoarctation have been reported[1,3–11,13–20] and 9 of these had other associated congenital cardiac anomalies.[7,10,16,17,18] This does not necessarily imply an increased incidence of associated cardiac anomalies, inasmuch as the aortic kinking was an incidental finding and was detected only because of the angiographic study prompted by the associated heart lesion.

Many of the published angiograms of kinked aorta demonstrate that the distance between the origin of the left common carotid and left subclavian arteries is greater than normal.[1,12] Barry's[2] description of the complex changes that occur in the embryonic aorta may explain such developmental abnormalities. In organogenesis, some arterial segments elongate, whereas others become greatly contracted. One such change occurs in the 7th dorsal intersegmental arteries when they undergo a cephalad shift and become the origin of the subclavian arteries. When the heart descends into the thorax, the subclavian artery remains relatively cephalad. There is associated compression of the 3rd to 7th segments of the dorsal aortic roots and the 4th aortic arch segments. Failure to compress the left 4th aortic arch and the 3rd to 7th dorsal aortic root segments during development results in an abnormally long aortic arch that twists at the point of insertion of the ligamentum arteriosum.

In the previously reported cases, no symptoms have been ascribed to the aortic

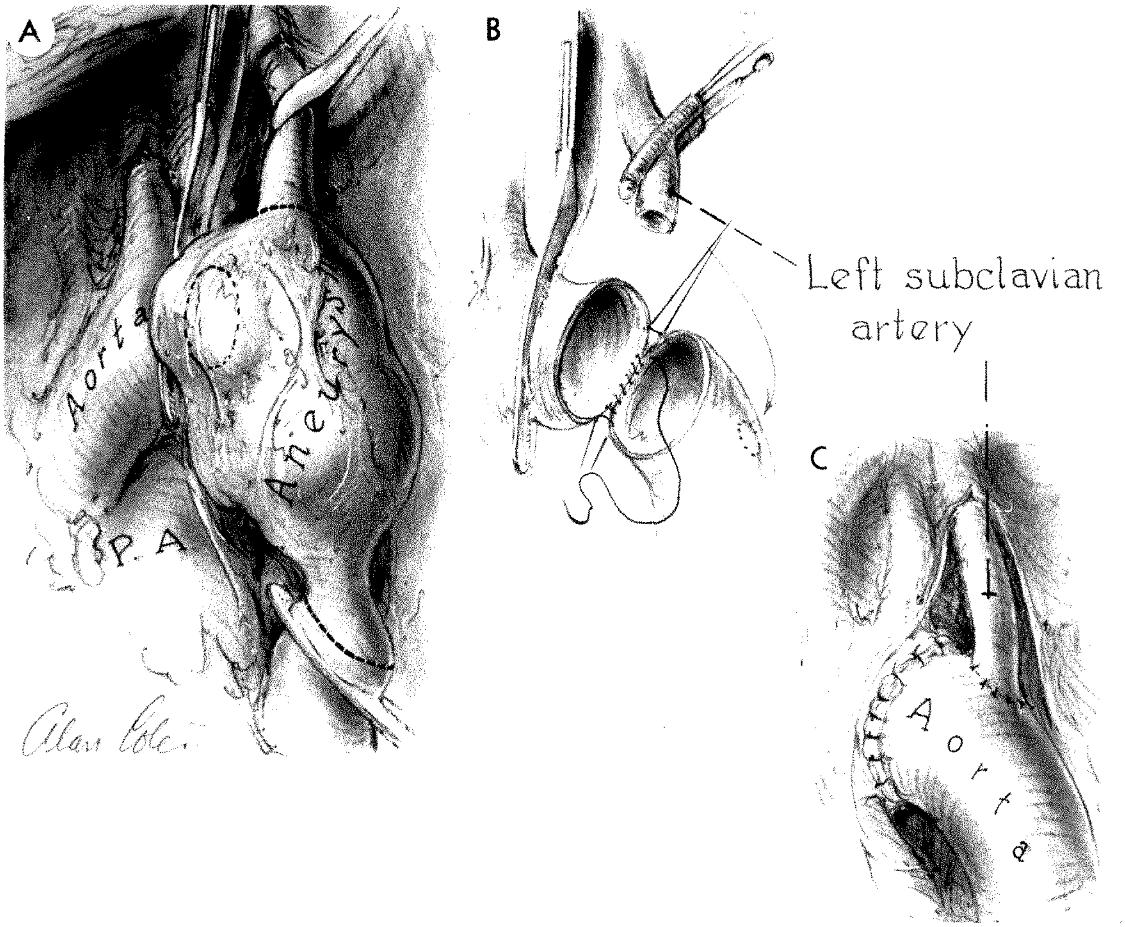

FIG. 6. (*A*) Sketch showing lobulated aneurysm proximal to ligamentum arteriosum. (*B*) Clamping of left subclavian artery and excision of aneurysm. (*C*) Anastomosis of aorta with left subclavian artery reimplanted into aorta.

anomaly. Several patients, however, have had murmurs which Bruwer and Burchell[4] attributed to hemic turbulence through the kinked arch of the aorta. In Steinberg's[16] series associated aortic valvular malformations were also believed to be factors in the development of such murmurs. In our case, the prominent systolic murmur which was present for 6 years was thought to be due to turbulence. This turbulence may also be a causative factor in the development of the lobulated aneurysm. Although dilatation of the ascending aorta and aneurysm of the aortic sinuses have been reported with pseudocoarctation,[1,10,18] to our knowledge aneurysm formation at the site of aortic kinking has not been previously reported. Šarić *et al.*[14] in 1960 postulated that such a complication could occur. They pointed out that pathophysiologically, the anomaly was similar to an aneurysm. Instead of the usual arch, there exist three curves which sustain the full impact of the blood stream that pounds against the bends and produces an enlargement. As long as the wall remains elastic, there is no danger of aneurysm formation. As the wall becomes less elastic, the lateral pressure increases at the site of convexity and the artery widens further, causing more damage to its wall. Hence, an aneurysm appears in the parts most exposed to the blood stream. Kinking of the aorta, once suspected, is easy to prove with angiography.

The differentiation of this anomaly from coarctation of the aorta depends clinically upon the demonstration of normal pulses and blood pressure in the lower extremities and angiographically the absence of collateral flow via the intercostal and internal mammary arteries. The importance of proper roentgenographic studies in the demonstration of pseudocoarctation has been stressed.[15] Berry *et al.*[3] reported a case explored surgically on the basis of a venous angiogram done in the posterior anterior position. In this view, the lesion was mistaken for a vascular aneurysm of the aorta. In the case presented, angiograms were taken in both the left anterior oblique and right anterior oblique positions, giving the best evaluation of aortic anomaly and aneurysm structure.

SUMMARY

Pseudocoarctation of the aorta is a rare anomaly. Until now, it has been considered to be a benign condition. The case presented in this paper was followed for 12 years, at which time a trilobulated aneurysm developed at the site of the aortic kink. To our knowledge, this is the first reported case of such a complication. A possible reason for the aneurysm formation is suggested and the differentiation of pseudocoarctation from true coarctation is briefly discussed.

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PAINLESS CHRONIC DISSECTING ANEURYSM OF THE AORTA*

REPORT OF A CASE

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SYNCOPE, rather than chest or back pain, initiated the onset of dissection of the aorta in a 57 year old woman. A year later, following another mild syncopal attack in church on a very hot summer day, re-examination disclosed enlargement of the ascending aorta which was unchanged over a year's observation. A systolic murmur and thrill over the base of the heart radiating into the neck suggested the diagnosis of aortic stenosis. For this reason, aortography was performed and established the diagnosis of chronic dissecting aneurysm of the aorta beginning at the root of the aorta and extending into the common iliac arteries. Because of the relatively asymptomatic status of the patient, surgical treatment was withheld. This case is reported in order to emphasize that aortic dissection may be painless, well tolerated, and be found incidentally.

REPORT OF A CASE

A 57 year old Caucasian housewife (N.Y.H. No. 1029141) was referred by Dr. Otto Baum for aortography on September 26, 1965. On June 1, 1964, while at the checkout counter of a supermarket, she suddenly lost consciousness, fell to the floor without injuring herself, and after a brief interval, revived. She had no pain and was able to stagger to her car in an adjacent parking lot. Because she felt "shaky," she was admitted to a hospital. She experienced only chilly sensations. Subsequently, she learned that she had normal blood pressure and a mild fever, which after a few days became normal. Penicillin via intravenous drip was given for 3 weeks even though blood cultures yielded no growth. The patient was discharged after 10 weeks and sometime later she had episodes of palpitation which were treated with quinidine. She was well until July, 1965, when she momentarily fainted. Examination and roentgen study of the chest led to her admission to this hospital. The patient had always been well except for mild hypertension which was discovered during a routine examination in 1960.

Examination revealed a well developed and well nourished woman resting comfortably. Her weight was 133 pounds (60.6 kg.), height, 55 inches. The apex of the heart was located at the sixth intercostal space, 2 cm. from the left midclavicular line. A basal systolic ejection (Grade 4–6) murmur radiating to the neck and along both sides of the spine in back was heard. A systolic thrill was also felt over the left carotid artery. The blood pressure readings were as follows: right arm, 120/80; left arm, 140/70; right leg, 220/150; and left leg, 200/130 mm. Hg. The pulses were equal. The lungs were clear, the liver was not enlarged, and neurologic examination was normal. A bruit was also heard in the abdomen.

Urine and blood studies were normal except for urea clearance of 45 per cent of normal. The electrocardiogram showed normal sinus rhythm, rate 85 per minute, some T wave abnormalities, and slight prolongation of the QT interval. Roentgen examination of the heart showed slight enlargement of the left ventricle with dilatation and enlargement of the ascending aorta (Fig. 1, *A* and *B*). Percutaneous right transfemoral supravalvular aortography and abdominal aortography disclosed dissection of the aorta beginning above the aortic valves and involving the entire thoracic and abdominal aorta and both common iliac arteries. The brachiocephalic arteries were uninvolved except at their origins, but the renal arterial and celiac axis circulations appeared altered (Fig. 2, *A–D;* and 3, *A* and *B*).

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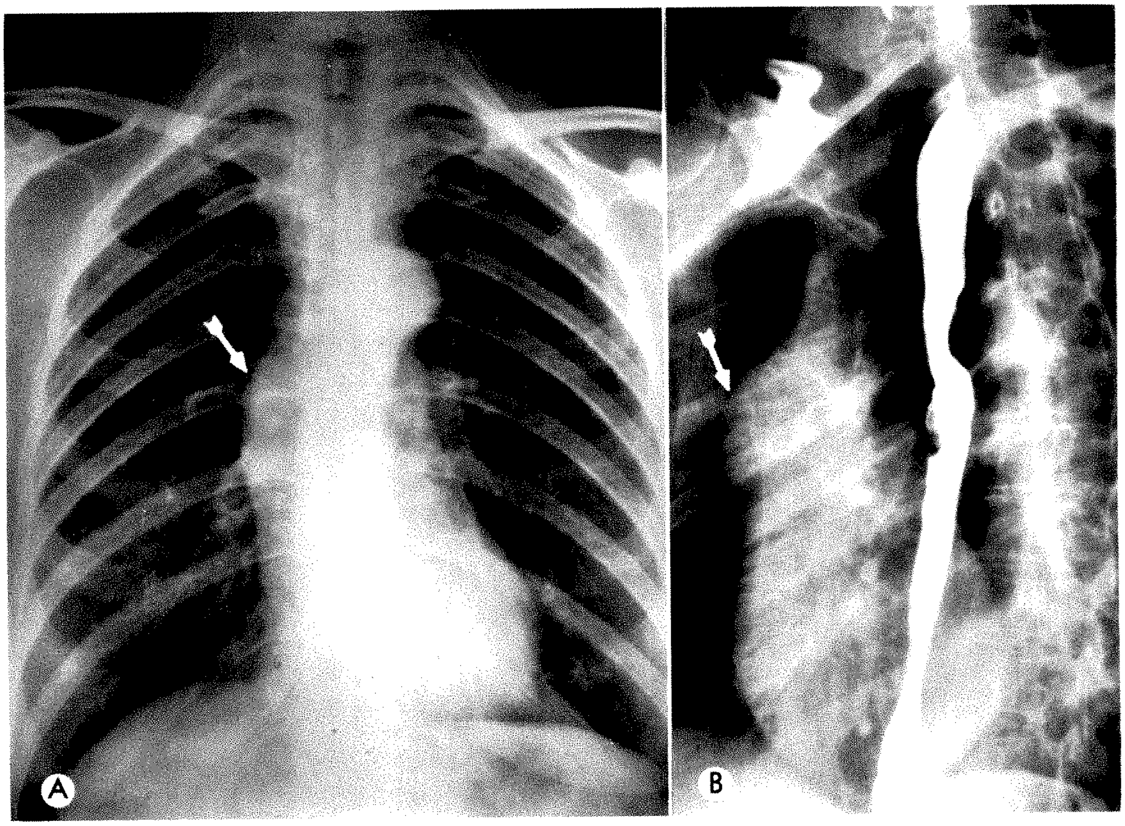
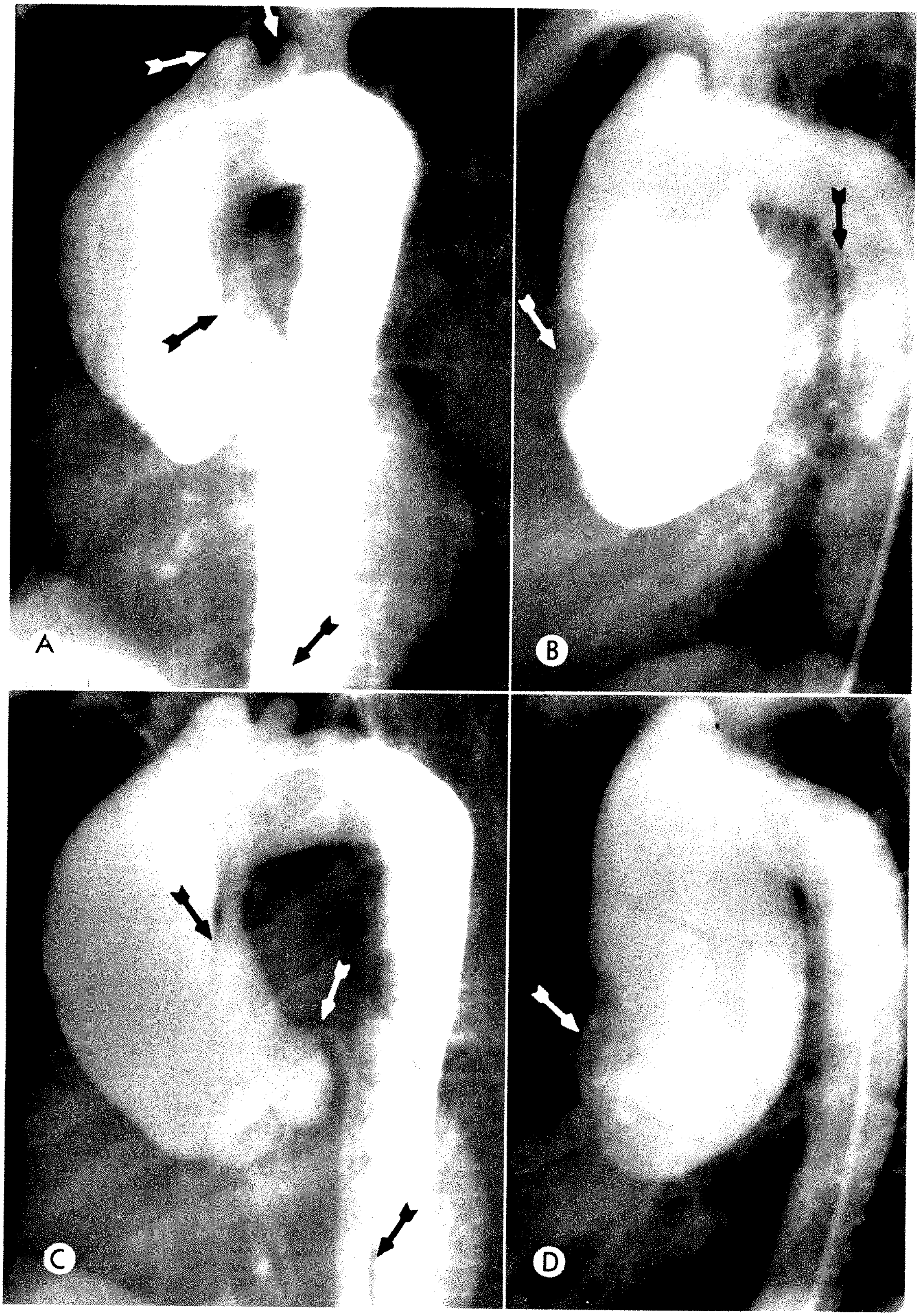


FIG. 1. (*A*) Frontal teleroentgenogram showing slight enlargement of the left ventricle and moderate bulging of the ascending aorta (arrow). (*B*) Left anterior oblique esophagram showing the slightly enlarged left ventricle and prominent ascending aorta (arrow) to advantage.

DISCUSSION

Sudden onset of pain in the chest and back are classic symptoms of acute aortic dissection. The incidence of painless dissection has been estimated from 15 per cent[5] to 55 per cent.[1] Because of sudden death, loss of consciousness, confusion, and dysarthria, a record of pain may not be obtained.[6] It has been estimated that without treatment approximately 25 per cent of patients survive acute aortic dissection for more than 2 weeks, and approximately 10 per cent live for more than 6 months.[5] Recently, however, Wheat and associates[8] have pointed out that attempts at surgical repair in the acute stage of aortic dissection are often fatal. Rather than immediate surgical treatment,[3] they advocated treatment with antihypertensive drugs during the acute stage and surgery, only if indicated, when the patient improved. With this regimen survival occurred more often. As further proof of the benefit of their conservative treatment, they cited the prevention of spontaneous dissection of the aorta in young turkeys when fed small doses of reserpine. One of the striking achievements of their hypertensive treatment was alleviation of chest pain which previously had required large doses of narcotics.

Our patient was apparently normotensive when she was first hospitalized, and at this admission she had only minimal hypertension, 150/90 mm. Hg. Perhaps the syncope at the onset of the aortic dissection masked the pain; the relative hypotension following it may have prevented its recurrence. Although the enlargement of the ascending aortic shadow at first suggested aortic dissection, when she was seen at this center, her general well being and the aortic systolic murmur and thrill mitigated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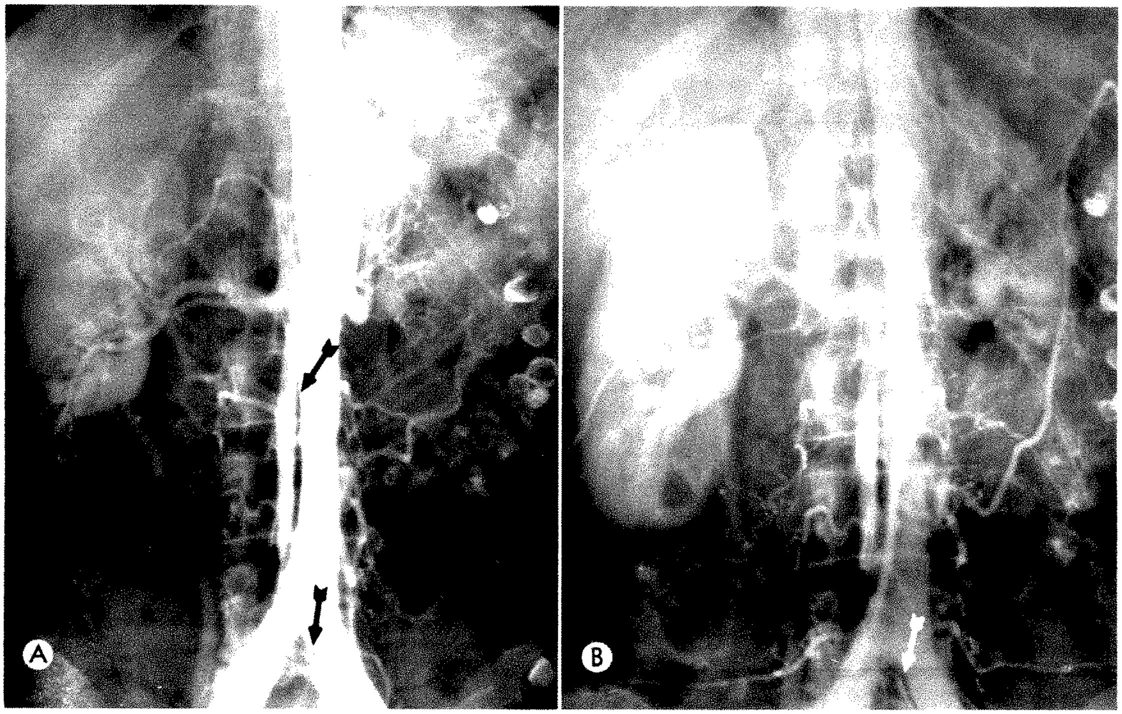

Fig. 3. (*A*) Selective abdominal aortogram showing the double-barrelled aorta and intimal layer between them (upper black arrow). The right renal artery was visualized from both the false and true channels. Only the upper pole of the left renal artery was visualized coming from the false channel. The dissection extends into the common iliac arteries (lower black arrow). The celiac axis is only partially visualized from the false channel, while the superior and inferior mesenteric arteries were not opacified. (*B*) Abdominal aortogram, later in the series, showing nephrographic phase of kidneys. The lower half of the left kidney is unopacified. Again, note line of dissection (white arrow).

in favor of the diagnosis of aortic stenosis. This seemed probable because there was no change in the conventional roentgenogram (Fig. 1, *A* and *B*) during a 1 year period. It was only after aortography (Fig. 2, *A*–*D;* and 3, *A* and *B*) that the true state of affairs was recognized.[2]

The patient exhibited none of the stigmata of the Marfan syndrome,[7] and so medial degeneration due to this disease was

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Fig. 2. (*A*) Off frontal selective aortogram with the catheter apparently in false channel showing the double-barrelled aorta. Black arrows point to the dislodged intimal layers of the ascending and descending portions of the aorta.[2] Note that the innominate and left common carotid arteries are dissected only at their origins (white arrows). (*B*) Biplane lateral view of *A*. Note point of communication (jet) between the true and false lumen (white arrow) and displaced linear intimal layer (black arrow). (*C*) Off frontal selective aortogram, at a later time, showing the left coronary artery originating from false channel (white arrow) and the intimal layer between the dissection (black arrows). (*D*) Biplane lateral view of *C* Again, note jet at site of communication of dissection (white arrow).

discounted. Indeed, the only significant predisposing condition was the history of hypertension. It would appear that the signs of aortic stenosis were due to the double-barrel deformity of the dissection at the aortic root; the thrill and the murmurs at the neck were apparently due to extension of the dissection to the origin of the great vessels causing some stenosis of their lumen (Fig. 2, *A–D*). The bruit in the abdomen was apparently due to the double-barrelled abdominal aortic dissection formation (Fig. 3, *A* and *B*).

During aortography, the catheter was apparently located in the false channel and, therefore, visualization of the aortic root failed to disclose both coronary arteries which were uninvolved in the dissection. The left coronary arterial circulation, partially visualized in Figure 2*C*, apparently was connected to the original channel. The right kidney and the upper half of the left kidney were seemingly fed by blood from the false aortic channel (Fig. 3, *A* and *B*). Failure to visualize the mesenteric arteries does not mean that the circulation to these vessels was occluded. Rather, they could have been fed by blood from the true aortic channel. For complete proof of this, percutaneous retrograde left femoral arteriography could have been performed in order to demonstrate the true lumen of the aorta. Since this maneuver seemed to be of academic interest rather than of practical importance, re-examination was not attempted. Despite the extension of the dissection into the iliac arteries, inequalities of the pulses and intermittent claudications of the lower legs were not present. The upper extremities' pulses were also not affected, probably because the dissection did not extend beyond the origins of the great arterial vessels (Fig. 2, *A–D*).

Classically, regurgitant aortic murmurs are common following aortic dissection. Indeed, the incidence has ranged from 20 per cent[5] to 56 per cent.[1] The aortic systolic murmur in our patient was, therefore, unique, and was due to aortic deformity caused by the dissection. The causes of pain in aortic dissection are not certain. O'Donovan *et al.*[6] favor the theory of Wood, Pendergrass, and Ostrum,[9] who state that pain is prevented by the dissecting hematoma bulging the intima inward, by early re-entry into the aortic lumen preventing ischemia, and by slow dissection preventing sudden violent distention of the adventitia.

SUMMARY AND CONCLUSIONS

Painless aortic dissection involving the aorta from above the aortic valves extending into the common iliac arteries was demonstrated in a 57 year old woman whose only complaint was mild syncope. Over a 1 year period, the conventional roentgenogram of the chest showed only moderate ascending aortic dilatation. This, together with a systolic murmur and a thrill at the base of the heart radiating into the neck, suggested aortic stenosis rather than dissection. Because the patient was normotensive and asymptomatic, conservative treatment, medical rather than surgical, was prescribed. This case emphasizes that aortic dissection may occur without pain and should be suspected whenever unexplained cardiac murmurs and aortic dilatation develop.

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UNSUSPECTED DISSECTING HEMATOMA (ANEURYSM) AND RIGHT CORONARY ARTERIAL OCCLUSION FOLLOWING OPEN-HEART MITRAL VALVULOTOMY*

REPORT OF A CASE

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WHEN aortic dissection (hematoma) follows femoral artery cannulation during open-heart surgery, it is usually fatal.[1,3,7] In contrast to this, is the case herein reported of a woman who survived hemothorax, shock, and cardiac arrhythmias which followed the open-heart surgery; aortic dissection due to femoral arterial cannulation and coronary occlusion were found unexpectedly 8 months later during left ventriculography performed in order to assess the degree of mitral insufficiency. Thus, not all aortic dissections following femoral arterial cannulation during open-heart surgery are fatal.

REPORT OF A CASE

A 36 year old Caucasian woman (N.Y.H. No. 621522) was first admitted to The New York Hospital on May 15, 1952, complaining of dyspnea on exertion of 6 months' duration. Rheumatic heart disease had been diagnosed in 1943, but she was unaware of fever, rheumatism, sore throats, or chorea. Three months prior to admission, in February 1952, she had had weakness of the left arm and leg and this had gradually subsided. On admission in 1952, the heart was enlarged, the rate was regular, and the blood pressure was 118/80 mm. Hg. An apical diastolic thrill, a crescendo presystolic apical murmur, and short harsh systolic and diastolic murmurs of mitral stenosis were heard over the precordium. The liver edge was palpable but there was no edema of the legs. The electrocardiogram showed regular sinus rhythm, sinus brachycardia, right axis deviation, and high P waves. Cardiac catheterization by Dr. Daniel S. Lukas on May 16, 1952, showed pulmonary and right ventricular hypertension (45/19 and 45/7 mm. Hg, respectively), increased pulmonary wedge pressure, and low cardiac indices—all of which worsened after exercise. The mitral orifice was calculated to be 1.1 cm.^{2}.

Shortly after admission in 1952, she complained of pain in the left lower lateral and posterior chest; rales were elicited in this area and the roentgenogram of the chest (Fig. 1 *A*) showed parenchymal haziness with obliteration of the costophrenic sulcus consistent with pulmonary embolism and infarction. On May 29, 1952, closed mitral valvulotomy via the left atrial appendage route was performed, and with satisfactory enlargement of the mitral orifice. The left atrial appendage contained mural thrombus and this was affirmed by histologic study of the excised appendage. She improved, and was discharged on June 12, 1952. A follow-up examination 9 years later, on October 6, 1961, showed enlargement of the heart and pulmonary arterial system. The lungs were clear (Fig. 1 *B*).

On September 11, 1963, 11 years later, after a long period of improvement, she was readmitted because of sudden onset of numbness, coldness, and purplish discoloration of the left lower extremity. She had had bouts of palpitation during a period of several months and this had been controlled by quinidine. Rales were heard at the bases. The heart was irregularly irregular (atrial fibrillation). The second pulmonic sound was loud and split, and holosystolic (Grade 1) and diastolic (Grade 1) apical murmurs were elicited. There was edema (1+) of the extremities. Cardiac catheterization on October 2, 1963, again revealed right ventricular and pulmonary arterial hypertension which increased following exercise. The mitral valve area was

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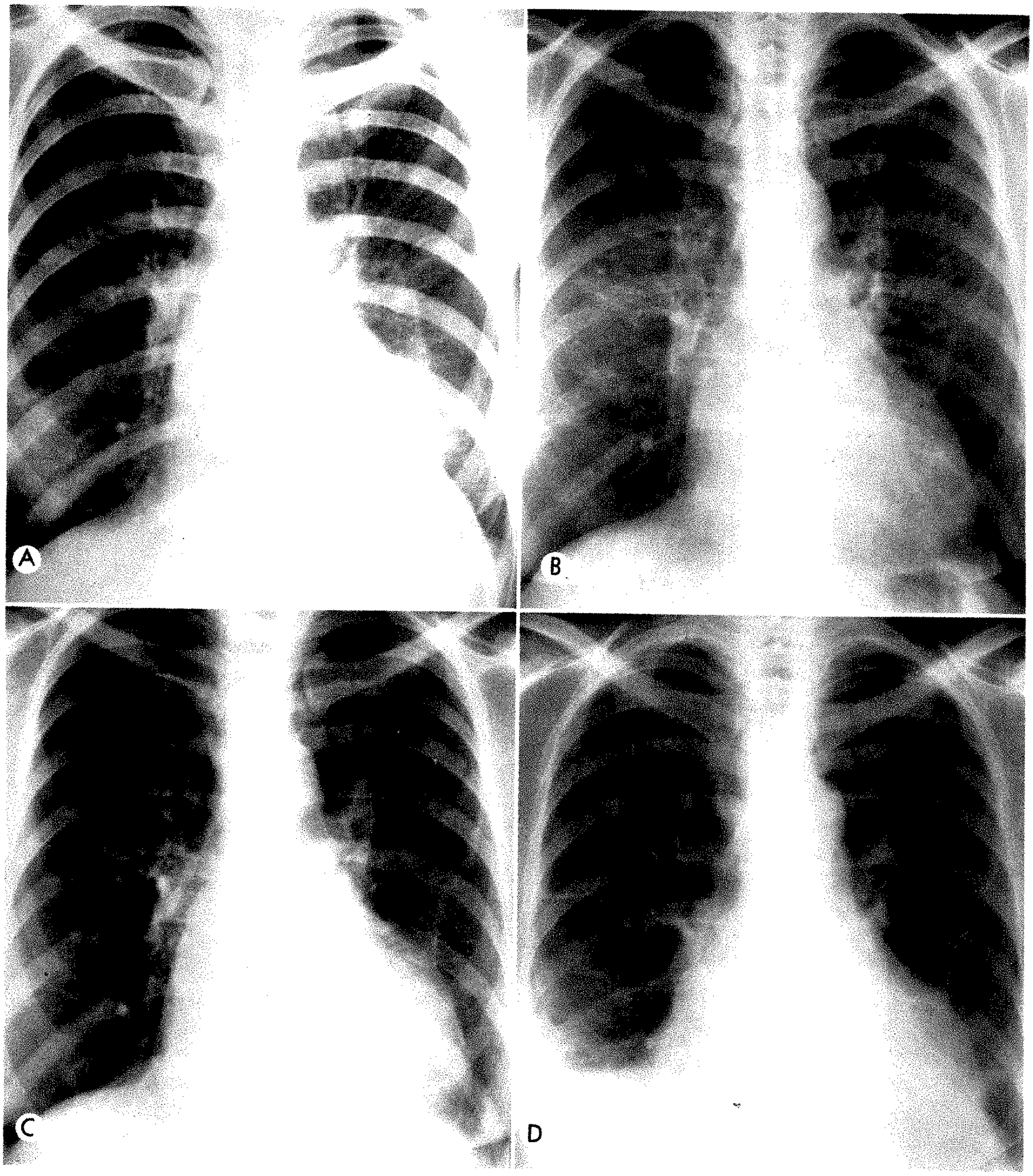

Fig. 1. *Serial frontal conventional roentgenograms of the chest in severe mitral valvular disease.* (*A*) Teleroentgenogram, made on May 12, 1952, showing enlargement of the heart and pulmonary artery, plethoric lungs, and pulmonary densities near the apex of the heart and left costophrenic sulcus. (*B*) Frontal chest roentgenogram, made 9 years later, showing clear lung fields and enlarged heart and pulmonary arterial system. (*C*) Frontal teleroentgenogram of the chest, November 9, 1963, disclosing enlargement of the heart and pulmonary artery. (*D*) Frontal teleroentgenogram, June 1964, showing fluid at right base of the lung.

calculated to be 0.8 cm.^{2} and tricuspid insufficiency was also demonstrated. A roentgenogram of the chest taken on November 9, 1963, showed further enlargement of the heart (Fig. 1 *C*). She improved after the dosage of digitalis and mercurial diuretics were increased. On November 11, 1963, open-heart mitral valvulotomy was performed using a rotating disk type pump oxygenator. Both common femoral arteries were exposed and tapes were passed about them so that No. 16 plastic catheters and 5 mm. metal cannulas could be inserted in the proximal end of the vessels for return of arterialized blood from the artificial heart-lung machine. A soft, recent, fresh thrombus measuring $3.5 \times 1 \times 0.5$ cm. was removed from the posterior wall of the left atrium. The mitral orifice measured approximately 0.8 $cm.^2$ and contained a small amount of calcification. The Gerbode dilator was used to enlarge the orifice to approximately 3.0 $cm.^2$. Some mitral regurgitation at the site of split of the posterior commissure was seen but the degree of insufficiency did not appear to be significant. The total time on the artificial heart-lung machine was 33 minutes. The catheters and cannulas were removed from the femoral arteries which appeared normal and these were sutured with No. 00000 silk.

Following surgery, atrial fibrillation, atrial flutter with 2:1 block, and shock (90/50 mm. Hg) developed. Roentgenography of the chest revealed left hydrothorax, and thoracentesis yielded 270 ml. of bloody fluid. On the following day, November 14, 1963, dyspnea was pronounced and 2,400 ml. of bloody fluid was aspirated from the left hemothorax. Further improvement followed transfusion and cedilanid therapy and permitted her to lie flat in bed. Ambulation began on the sixth day postoperatively, and quinidine sulfate (1.2 gm.) changed the atrial fibrillation to regular sinus rhythm. She was discharged on December 3, 1963.

The patient was readmitted on June 16, 1964, because of congestive heart failure. The neck veins were distended, both lung bases were dull and contained moist rales, the extremities were markedly edematous, and the liver was tender and enlarged 10 cm. below the right costal margin. The venous pressure was elevated (160 mm. H_2O), the heart was enlarged, and apical diastolic (Grade IV) and blowing systolic (Grade III) murmurs were heard. No aortic valvular murmur was elicited. The rate was 100 per minute and irregular (atrial fibrillation); the blood pressure was 126/80 mm. Hg. The roentgenogram of the chest showed enlargement of the heart and congestion at both bases (Fig. 1 *D*). The electrocardiogram showed atrial fibrillation with premature ventricular contractions.

Percutaneous transaxillary left ventriculography on July 20, 1964, showed moderate to marked mitral insufficiency and dissecting hematoma (aneurysm) below the origin of the left subclavian artery (Fig. 2, *A* and *B*). Supravalvular aortography disclosed mild aortic insufficiency and occlusion of the right coronary artery at its origin (Fig. 3, *A* and *B;* and 4, *A* and *B*). Following bed rest, diuretics, and readjustment of the dosages of digitalis, the rhythm became regular. She improved and was discharged on July 21, 1964.

The patient became worse and was readmitted to the hospital in severe congestive heart failure on March 25, 1965. Her weight had increased from 45 to 59 kg. despite vigorous diuretic therapy. She also had developed a generalized dermatitis and a lupus erythematosus test was positive. The electrocardiogram showed atrial fibrillation, atrioventricular dissociation, and nodal tachycardia. The rate was 140 per minute; the atrioventricular dissociation, nodal pacemaker and nodal pauses were believed to be due to digitalis toxicity. The changes of an old diaphragmatic myocardial infarction (Q waves in leads 2, 3, and AVF) were also present. The congestive failure and dermatitis improved after adjustment of the dosage of digitalis, and discontinuation of some of the diuretics. She was discharged on April 12, 1965, and since then she has attended the cardiac clinic regularly. Weight gain, enlargement of the liver, and considerable edema of the legs and ascites persisted. Atrial fibrillation at a rate of 90 per minute was present.

The patient was re-admitted for heart failure and, following improvement, was transferred to a convalescent nursing home, where she died on January 31, 1966. Permission for postmortem examination was not given.

DISCUSSION

Jones *et al.*,[3] in 1960, apparently were the first to report fatalities in 2 patients following femoral arterial cannulation during open-heart surgery for alleviation of rheumatic mitral and aortic valvular disease. Cooley and colleagues[1] described 1 death from dissecting aneurysm in 37 cases of cardiopulmonary bypass following retrograde femoral arterial perfusion for left ventricular aneurysmectomy. Williams and Johnson[7] reported the death of a patient following aortic dissection after femoral cannulation for prosthetic replacement of

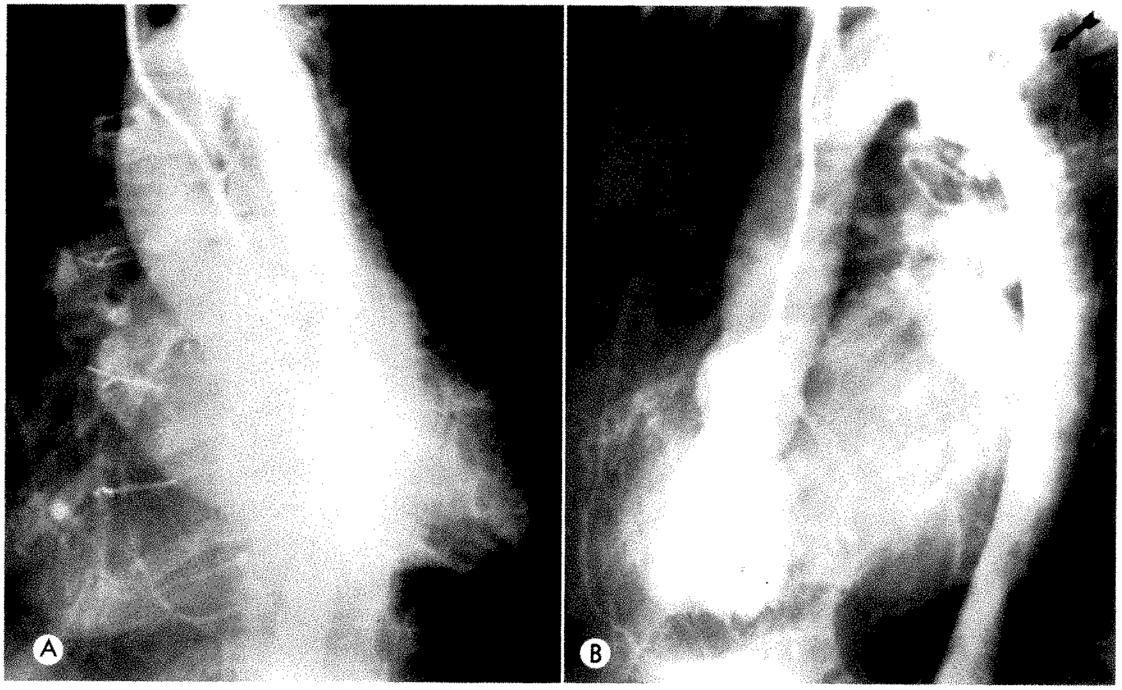

Fig. 2. *Selective left ventriculograms.* (*A*) Frontal view showing severe mitral regurgitation and aortic dissection below the origin of the left subclavian artery. (*B*) Biplane lateral view of *A* also showing the mitral regurgitation and aortic dissection (arrow).

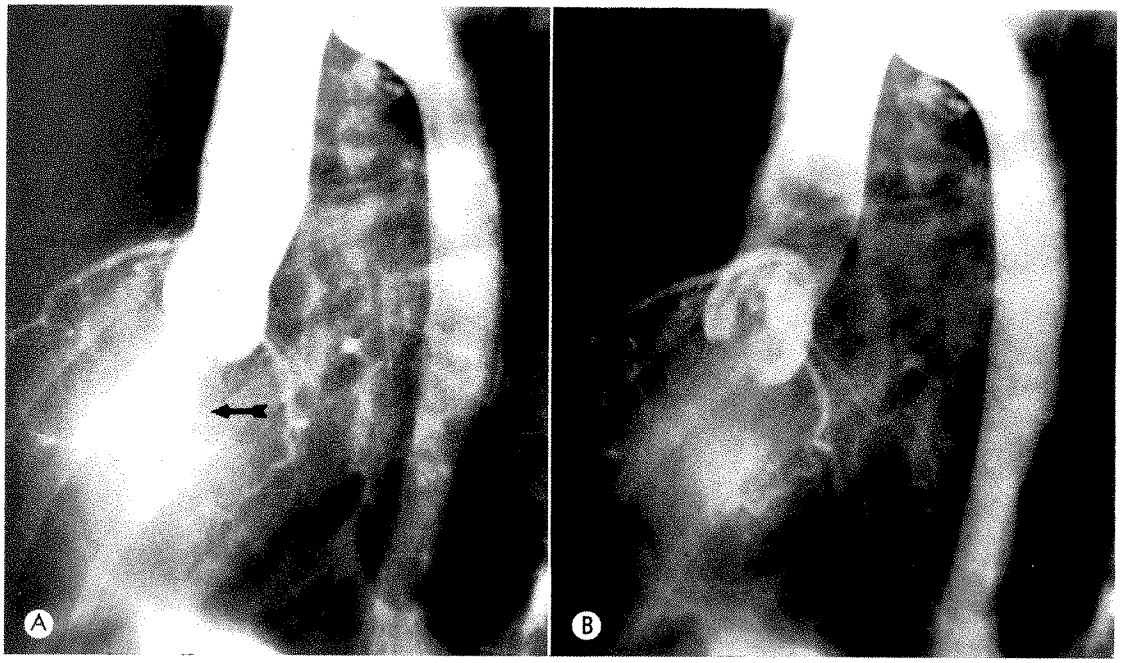

Fig. 3. *Supravalvular selective aortograms.* (*A*) Lateral aortogram, made during ventricular diastole, showing mild aortic regurgitation (arrow). (*B*) During systole of the heart, the right coronary artery is not visible, the left anterior descending and circumflex coronary arteries are well visualized.

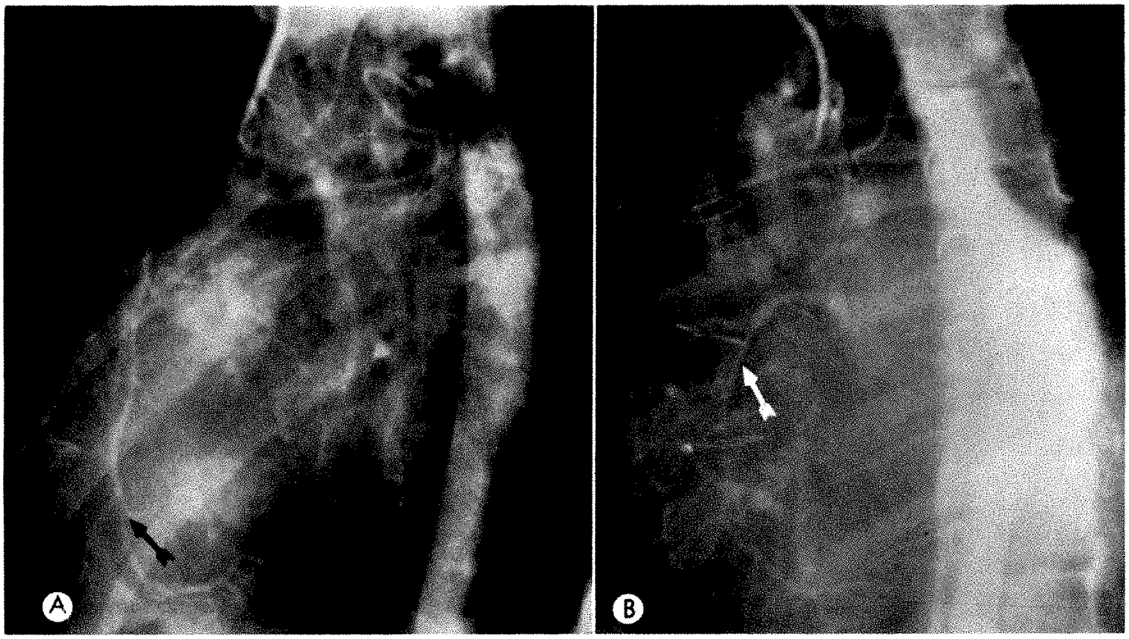

Fig. 4. *Later coronary arteriograms.* (*A*) Lateral coronary arteriogram of the right coronary artery opacified in retrograde fashion (arrow). (*B*) Simultaneous, biplane, off-frontal view of *A*, also showing the right coronary artery opacification by retrograde flow from the opposite left coronary arteries.

the aortic valve. Another fatality following open-heart surgery and femoral arterial cannulation during aortic valvular replacement with a prosthesis also occurred in this center, and has not as yet been published. Elliot and Roe[2] reported 2 fatal cases following aortic dissection during cardiopulmonary bypass. In the discussion of their paper, Dr. William Kerth of The Presbyterian Hospital, San Francisco, reported on 3 cases, 2 of which were fatal. Dr. Lyman A. Brewer, III, of Los Angeles also reported 2 cases with 1 death. Finally, Dr. William W. L. Glenn added 2 cases with 1 death. This suggests that the incidence of this complication is higher than hitherto assumed.

That dissecting hematomas (aneurysms) may become chronic and not necessarily fatal is demonstrated in the case reported above and by the other cases reported from this center in 1951.[4] Classically, pain in the chest and back is the presenting symptom of aortic dissection. O'Donovan *et al.*,[5] however, reported a patient with aortic dissection who was without pain. In the patient reported above, the left hemothorax and shock following open-heart surgery was at first believed to be responsible for the thoracic discomfort. The arrhythmia was also attributed to the operative procedure. Subsequent aortographic studies demonstrating the right coronary arterial occlusion and aortic dissection, indicate that these events were probably largely responsible for the postoperative arrhythmia and collapse. Recovery following thoracentesis and blood replacement suggests, however, that blood loss was also a factor in the patient's shock.

Repair of a dissecting aneurysm of the thoracic aorta following catheter aortography made via the left brachial and subclavian arterial routes was reported by Templeton *et al.*[6] It is conceivable that repair of an aortic dissection following femoral arterial cannulation during open-heart surgery is also feasible.[7] Such a procedure was contemplated following the diagnosis of dissecting aneurysm in the patient reported above, but the severe heart failure owing to mitral regurgitation made it imperative that the mitral valve be replaced by a prosthesis. This and the right coronary arterial occlusion are, however, formidable.

SUMMARY AND CONCLUSIONS

A dissecting hematoma (aneurysm) of the thoracic aorta below the left subclavian artery and significant occlusion of the right coronary artery were demonstrated in a woman being considered for mitral valve replacement following selective supra-aortic valvular aortography and left ventriculography. The femoral arteries had been cannulated during open-heart mitral valvulotomy 8 months earlier and may have been the cause of the aortic dissection since such complications have followed femoral arterial cannulation incident to open-heart surgery. Recovery following operation in our case was stormy and had been attributed to hemothorax, shock, and cardiac arrhythmia because she had improved following blood transfusions and restoration to normal sinus rhythm. The coronary arterial occlusion and aortic dissection were not detected until they were revealed by selective aortography. Care is necessary to avoid aortic dissection during femoral arterial cannulation for open-heart surgery. Coronary arterial occlusion is also a hazard of cardiac surgery, and apparently occurs when calcific plaques or thrombus are dislodged from either the valves or heart.

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REFLUX AORTOGRAPHY COMBINED WITH THE VALSALVA MANEUVER IN THE DIAGNOSIS OF ABDOMINAL AORTIC ANEURYSMS[*]

By MARVIN J. FRIEDENBERG, M.D., *and* TOM W. STAPLE, M.D.
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ANGIOGRAPHY is the most definitive method of diagnosing and preoperatively evaluating abdominal aortic aneurysms. The purpose of the authors is to describe a technique of abdominal aortography in which use of the Valsalva maneuver is combined with retrograde injection of contrast material through a cardiovascular needle in one common femoral artery.

METHOD

Under sterile conditions and local anesthesia, a 2¾ inch long, 15 gauge cardiovascular needle[*] is inserted percutaneously into a common femoral artery. A re-usable high pressure flexible nylon connecting tube[†] is attached to the needle. The tip of the needle is advanced up the lumen of the artery while a forceful and pulsating free flow of blood is constantly maintained in the connecting tube, in order to ensure proper positioning of the needle in the vessel. The connecting tube is then attached to an automatic pressure injector.[‡] The patient performs the Valsalva maneuver and, immediately upon its sudden release, 50 ml. of 60 per cent methylglucamine diatrizoate[§] is injected in about 1 second under 7–9 kg./cm.^{2} or 400 p.s.i. pressure. The needle must be held in place manually or with tape during the injection of contrast material to prevent recoil. Eight to 12 films are exposed in the anteroposterior projection at a rate of 2 per second, using the Schönander 14×14 inch or Franklin 10×12 inch rapid changer. Filming is begun at the start of the injection.

THE VALSALVA MANEUVER

Figure 1 is a pressure recording obtained with a catheter in the mid-abdominal aorta while a patient performs the Valsalva maneuver. At the onset of straining down against the closed glottis (arrow A), venous return to the lungs decreases and blood already in the lungs is forced into the systemic circulation. A temporary increase in pressure occurs in the abdominal aorta (arrow B). Because of the decrease in venous return to the lungs, however, cardiac output soon decreases, and this is reflected in a marked decrease in intra-aortic pressure (arrow C). Maximum reduction in intra-aortic pressure usually does not occur until the Valsalva maneuver is sustained for at least 8 seconds. Immediately following sudden release of the Valsalva effort (arrow D), blood from the systemic circulation rapidly fills the pulmonary vascular bed, and an additional precipitous decrease in intra-aortic pressure occurs (arrow E). Systolic pressure in the aorta at this time usually is 30–50 per cent of resting levels. Injection of contrast material is timed to coincide precisely with this maximum decrease in pressure. The intra-aortic pressure then "rebounds" to slightly above resting levels (arrow F) and gradually returns to the preinjection level (arrow G).

The patient is instructed and rehearsed in the performance of the Valsalva maneuver. With him straining down against the closed glottis, the examiner will say "1, 2, 3, relax." Upon the command "relax" he suddenly releases his breath and stops

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[*] Becton, Dickinson & Company, Rutherford, New Jersey, Catalogue #15112.

[†] Becton, Dickinson & Company, Rutherford, New Jersey, Catalogue #42-0024 through 42-0031.

[‡] Gidlund Injection Syringe, Elema-Schönander Company, Stockholm, Sweden; Cordis Injector, Cordis Corporation, Miami, Florida.

[§] E. R. Squibb & Sons, 745 Fifth Avenue, New York 23, New York.

[*] From the Edward Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, Missouri.

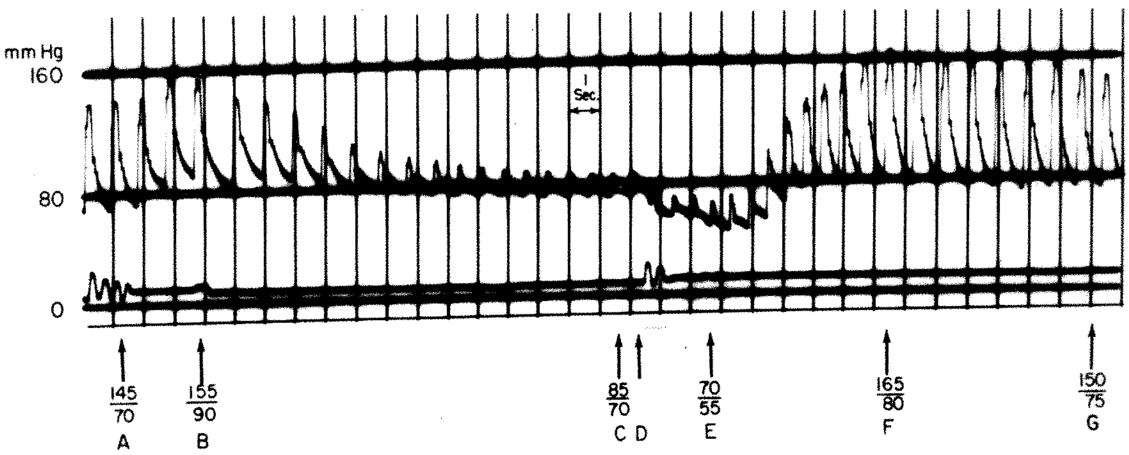

Fig. 1. Pressure recording in the mid-abdominal aorta with the patient performing the Valsalva maneuver. At the onset of straining down against the closed glottis (arrow A), the intra-aortic pressure increases and then gradually decreases over a period of about 8 to 10 seconds. Following sudden release of the Valsalva effort (arrow D), a precipitous decrease in pressure occurs during an approximately 2 second interval. Injection of contrast material is made to coincide exactly with this maximum decrease in intra-aortic pressure. After a slight rebound, the pressure gradually returns to normal.

straining. He is told not to breathe or move thereafter until filming is completed.

DISCUSSION

The diagnosis of abdominal aortic aneurysm frequently can be made by history and physical examination of the patient and the more routine abdominal roentgenographic procedures. Aortography, however, is useful for confirming the diagnosis and identifying those patients with an easily palpable or tortuous aorta which may simulate an aneurysm. We have examined 2 patients in whom aneurysms were suspected because of pulsatile abdominal masses, but aortography showed horseshoe kidneys and a normal aorta. Aortography also is useful in defining the size and extent of the aneurysm, including the degree of involvement of the renal and iliac arteries.

Each of the presently used angiographic techniques has undesirable features. Translumbar aortography[3] often is associated with complications.[7] The Amplatz[1] technique of translumbar aortography, using a polyethylene catheter does not completely avoid the hazard of puncture of the wrong artery or the weakened wall of an aneurysm. Use of a catheter advanced retrograde into the abdominal aorta from the femoral artery may be difficult or impossible because of marked tortuosity and arteriosclerosis of the iliac arteries and the distal aorta. The guide or catheter may dislodge a thrombus or possibly rupture the wall of the aneurysm. Intravenous[2,8] or right atrial[5] aortography has the advantage that contrast material is injected at a site remote from the aneurysm. However, dilution of contrast material limits detailed evaluation of abdominal vessels. Brachial or axillary aortography with or without a catheter[6] may be used, but these techniques involve either unnecessary dilution of the contrast material or the added risks incurred by introduction of a catheter. The aorta has also been opacified by bilateral simultaneous pressure injections through needles in both femoral arteries,[9] a relatively unwieldy technique which duplicates some effort. We have used the Valsalva maneuver for bilateral aorto-ilio-femoral arteriography with a manual injection of contrast material through a unilateral percutaneous common femoral artery needle.[4] However, a manual injection may incompletely opacify a large aneurysm and may fail to demonstrate its relationship to the renal arteries and the uninvolved proximal aorta.

We have performed retrograde pressure

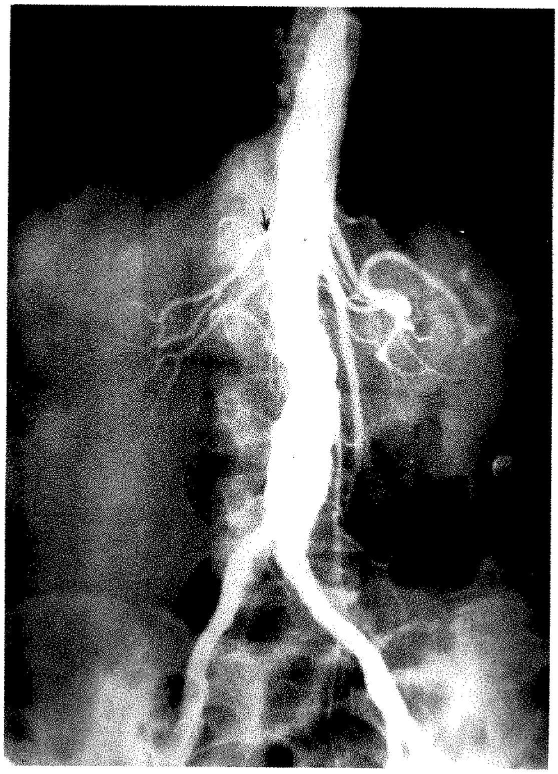

FIG. 2. Reflux aortogram through a left common femoral artery needle after sudden release of the Valsalva maneuver. Contrast material refluxes into the distal thoracic aorta and opacifies the celiac axis, renal arteries and mesenteric arteries. A small abdominal aortic aneurysm is opacified. A stenotic segment (arrow) is demonstrated in the more cephalad of 2 right renal arteries.

abdominal aortography by the technique described in over 100 patients without a complication (Fig. 2). Although in our experience the jet of contrast material never has dislodged an arteriosclerotic plaque, we recognize this theoretic possibility. Reflux of contrast material into the abdominal aorta above the aneurysm may be limited by extreme tortuosity of the iliac arteries (Fig. 3). Nevertheless, enough practical information is obtained by the examination in these occasional patients to warrant its use. In some cases a large aneurysm may be filled with blood clot, except for a smooth central channel (Fig. 4, *A* and *B*). The roentgenographic diagnosis depends upon recognition of calcific plaques in the intima considerably removed from the opacified central channel and a soft tissue mass at the site of the aneurysm. Patients with abdominal aortic aneurysms often have extremely arteriosclerotic and tortuous iliac arteries. Reflux aortography using the Valsalva maneuver has the advantage that a concentrated bolus of contrast material opacifies the abdominal aorta without requiring the use of a wire guide or catheter in patients in this high-risk group.

SUMMARY

A simple technique of reflux aortography for demonstration of adominal aortic aneurysms is described. The technique combines the use of the Valsalva maneuver with retrograde pressure injection of contrast material through a unilateral percu-

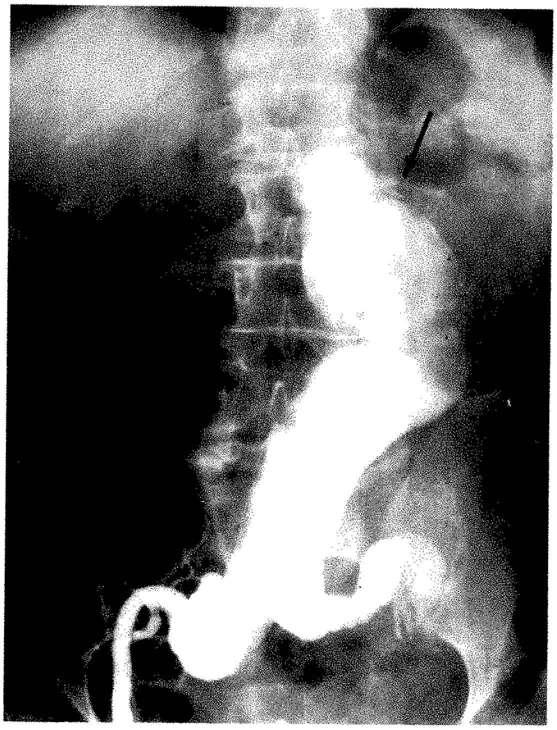

FIG. 3. Reflux aortogram through a right common femoral artery needle after sudden release of the Valsalva maneuver. The extremely tortuous iliac arteries prevent reflux of contrast material into the aorta proximal to the aneurysm, but considerable information is obtained by the examination. Both common iliac arteries are involved. The arrow indicates the left renal artery. Retrograde passage of a catheter through the pelvic vessels in this patient would have been difficult or impossible.

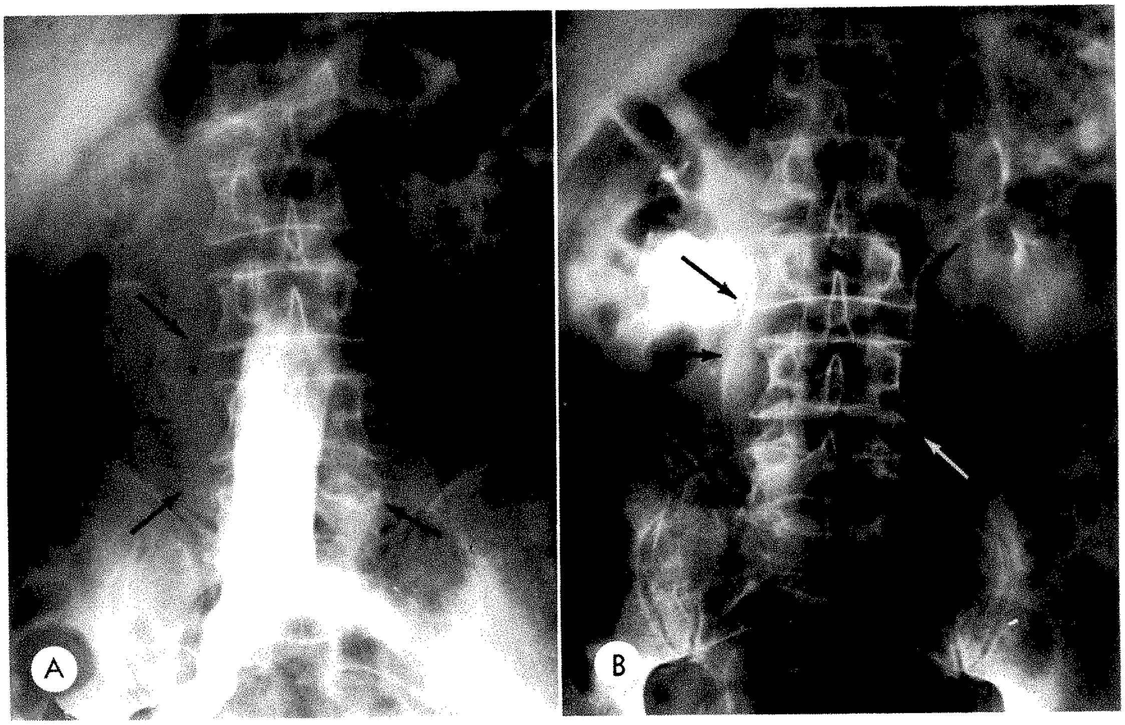

Fig. 4. (*A* and *B*) Reflux aortograms through a right common femoral artery needle after sudden release of the Valsalva maneuver. The central aortic channel is wide but smooth in outline. Blood clot fills the aneurysm from the central channel to the intima outlined by calcific plaques (arrows in *A*). The peripheral margins of the aneurysm are further delineated by anteroposterior roentgenograms of the abdomen (arrows in *B*) during intravenous urography.

taneous common femoral artery needle. It is particularly useful in the high-risk group of patients with abdominal aortic aneurysms, many of whom have extremely arteriosclerotic and tortuous iliac arteries which make passage of wire guides and catheters difficult and hazardous.

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SELECTIVE VISCERAL AND TOTAL ABDOMINAL ARTERIOGRAPHY VIA THE LEFT AXILLARY ARTERY IN THE OLDER AGE GROUP*

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PERCUTANEOUS transaxillary catheterization for arteriography has recently received considerable attention.[3,4,7] A variety of cardiac and neurologic lesions, particularly those involving the ascending and arch portions of the aorta, have been investigated by this route. The right axillary artery has been the portal of entry for most of these studies since anatomically this vessel yields ready access into the ascending aorta.

The left axillary artery may similarly be employed to investigate the descending and abdominal segments of the aorta. However, a technical problem arises in the older age group. In these patients the anatomic relationship between the left subclavian artery and aortic arch causes considerable difficulty in passing a catheter into the descending aorta (Fig. 1). Instead, in the elderly, the catheter is usually deflected into the ascending aorta. It is precisely though in this older age group, that the potential value of this approach is so useful, since this is the population afflicted with aortic occlusive disease,[2] aneurysms and femoral obstructive lesions, precluding femoral artery catheterization and making translumbar aortography extremely hazardous.

The purpose of the author is to describe a method which facilitates catheterization of the abdominal aorta from the left axillary artery when difficulty is encountered because of altered anatomic relationships. This approach permits selective arteriography of the branch vessels as well as total aortography and a few illustrative examples will be presented (Fig. 6 through 12).

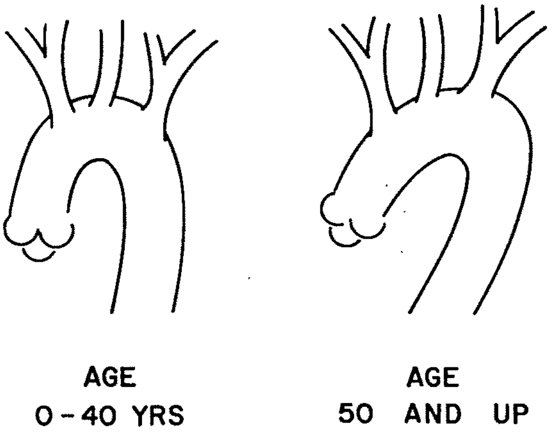

Fig. 1. Ectasia of the aortic arch in the older age group causes angulation between the left subclavian artery and descending aorta. As a result, catheterization of the descending aorta becomes technically difficult.

TECHNIQUE

The procedure requires the usual type of catheters and spring wire guide commonly used for the Seldinger technique. First, a red Ödman-Ledin, radiopaque catheter (2.2 mm. O.D.), approximately 65 cm. long is prepared with the tip tapered over a PE160 guide wire. The second catheter is a nonopaque PE240 (2.42 mm. O.D.), 70 cm. in length, with 8 side holes, and the tip also tapered to fit a PE160 guide wire. This size tapered tip is also suitable for occlusion by a tip occluder.[5] The PE160 spring guide wire is 120 cm. in length, with a very flexible end approximately 2.5 cm. long.

Prior to insertion of the red catheter, its end is molded into a hook by dipping it into boiling water. An important feature of this hook is that the length from the apex of the curve to the tapered tip be approximately 1.25 to 1.33 times the diameter of the aortic arch as judged on plain roentgenograms. In the majority of patients, this length measures 2.8 cm. to 3.5 cm. and suffices to prevent the catheter tip from slipping into the ascending aorta during the procedure (Fig. 2).

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The red opaque catheter is introduced first, and functions as the exploring catheter and when located in the abdominal aorta permits selective arteriography of the visceral branches as desired.

The left axillary artery is punctured, with the left arm abducted and the left hand comfortably placed under the patient's head. The elbow is then so adjusted as to best palpate the axillary pulse. The artery is punctured just under the pectoral fold, moderately high in the axilla, using the Seldinger technique[8] with a PE160 needle and guide wire. Image intensification is necessary to follow the guide wire through the axillary and subclavian arteries into the aortic arch. Occasionally, the guide wire is deflected into the long thoracic instead of the axillary artery. This is easily corrected by withdrawing the guide wire into the puncture needle and gently angulating the needle hub caudad and advancing the guide wire. The guide wire may also enter the left vertebral artery instead of passing down the subclavian artery into the aortic arch. Sometimes, simply torquing the guide wire will correct this problem. If not, when the red catheter with its hooked end is inserted, this difficulty is quickly resolved and the guide wire directed into the aortic arch.

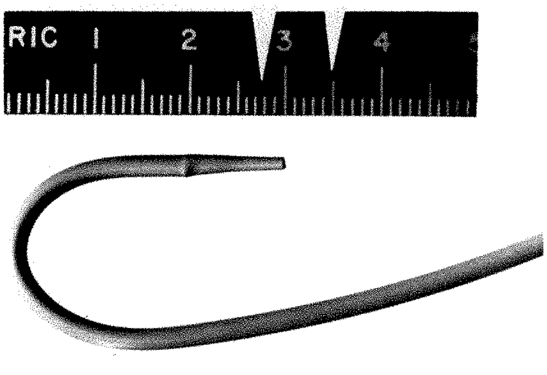

Fig. 2. The red opaque catheter is shaped into a U-type hook. The distance from the apex of the hook to tapered tip should measure approximately 2.8 to 3.5 cm. (arrows on ruler) depending on the aortic arch diameter.

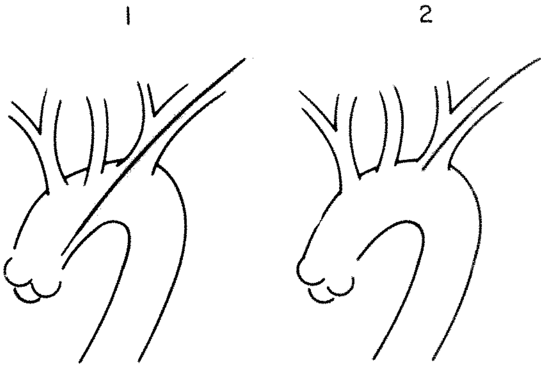

Fig. 3. Step 1. The red opaque catheter (stippled) is inserted over the guide wire (solid line) and enters the ascending aorta. Step 2. The guide wire is withdrawn into the catheter, allowing the pre-shaped hooked end to re-form in the aortic arch. The tip of the catheter points posteriorly with the patient in the right posterior oblique position.

If no difficulty has been encountered in entering the aortic arch with the guide wire and it is noted that the wire enters the descending aorta, then either the red opaque or PE240 catheter may be inserted. This depends on whether selective or total arteriography is indicated.

If instead, the guide wire enters the ascending aorta as most frequently occurs in the older age group, then the previously shaped red opaque catheter is inserted (Fig. 3). The wire is partially withdrawn so that the hooked end of the red catheter is re-formed in the aortic arch. The patient is then turned into the right posterior oblique position, a maneuver which opens the aortic arch curvature. The catheter tip is pointed posteriorly and the guide gently re-inserted.

Insertion of the guide wire controls the curvature of the hooked end of the catheter because the spring wire with its solid inner core (except for the distal 2.5 cm.) is stiffer than the catheter. Thus, by pushing on the guide wire and holding the catheter in place (Fig. 4), the hooked end assumes an ever widening arc directing the guide into the descending aorta. When the guide wire tip reaches the level of the diaphragm, both the catheter and wire are pushed forward simul-

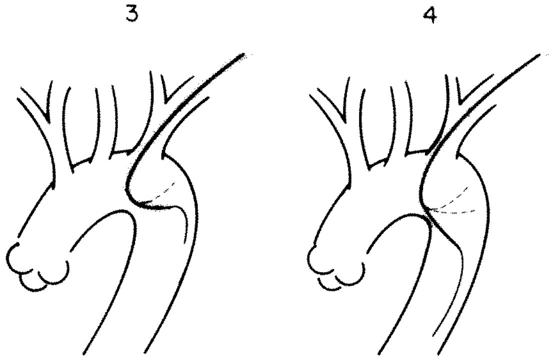

Fig. 4. Step 3. The guide wire controls the shape of the hooked end of the catheter. By gently inserting the guide wire and holding the catheter in place, the catheter straightens out. Step 4. The guide wire has been directed into the descending aorta, by the catheter, which has opened up in an ever widening arc. (Dotted lines represent successive positions in the widening arc.)

taneously (Fig. 5). As the catheter reaches the midabdominal aortic level, the wire is gradually withdrawn. This causes re-formation of the curved end, and facilitates selective catheterization of the visceral branches.

The curved end is also helpful in avoiding selective catheterization of the visceral arteries when only femoral arteriography is indicated. There appears to be a natural tendency for the guide wire to enter one of the visceral branches. By withdrawing the guide and re-forming the curved end in the abdominal aorta, the catheter easily slips by the visceral branch artery orifices.

For total aortography, the PE240 catheter with side holes replaces the red opaque catheter. This is accomplished by inserting the guide wire into the red catheter and maintaining the wire tip distal to the renal artery level while withdrawing the red catheter. This maneuver requires that the wire tip be observed under image intensification as the catheter is withdrawn. After complete withdrawal of the red catheter, the PE240 with side holes is inserted over the guide wire. Again the position of the wire tip is observed and it is necessary that the wire be gently withdrawn as the catheter is advanced, in order to maintain the position of the guide wire at the desired level. This maneuver is simply the reverse

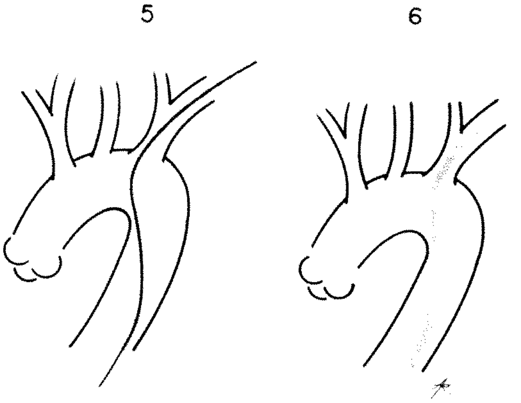

Fig. 5. Step 5. When the guide wire tip reaches the level of the diaphragm, the catheter and guide wire are advanced together. Step 6. The guide wire is withdrawn when the catheter tip reaches the desired position in the abdominal aorta or enters the particular visceral vessel selectively catheterized.

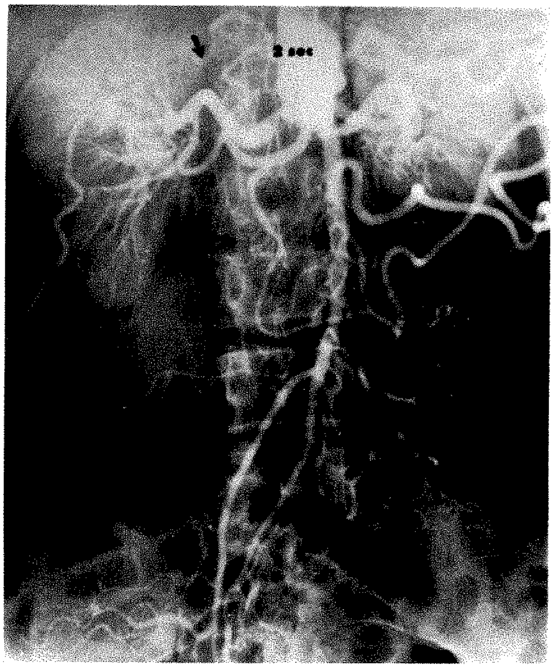

Fig. 6. A percutaneous left axillary aortogram in a 52 year old male with mild hypertension, minimal symptoms of claudication and absent femoral and leg pulses. Note the aortic occlusion and bilateral renal artery stenosis. The viscero-systemic collateral circulation is from the superior mesenteric artery via the dilated and tortuous middle colic artery (arrow head) and left colic artery (short arrow).

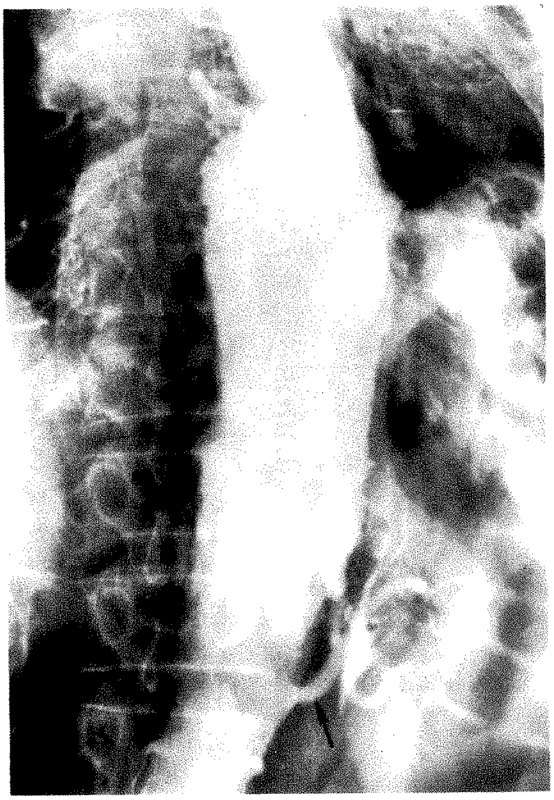

Fig. 8. A percutaneous left axillary aortogram in a 57 year old man with abdominal pain and a pulsatile mass. The fusiform shaped thoraco-abdominal aneurysm with its irregular contour is outlined. The left renal artery (arrow) appeared normal as did the right.

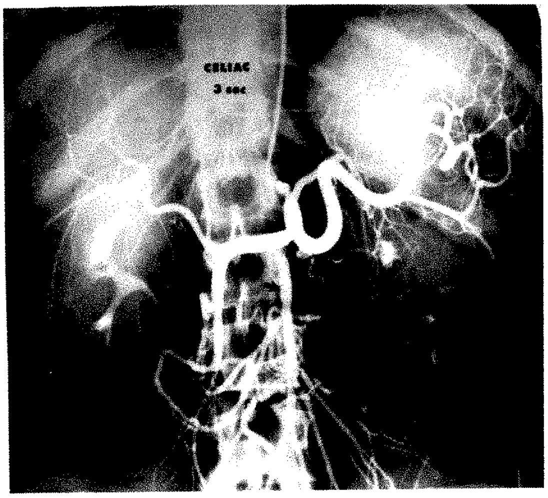

Fig. 7. Selective celiac arteriogram in a 44 year old premenopausal woman with hypertension, absent lower limb pulses, but no peripheral vascular or gastrointestinal symptoms. The celiac arteriogram demonstrates the partial celiac stenosis and collateral circulation via the pancreatic arcade vessels (thin arrows) to the occluded superior mesenteric artery (thick arrow). In addition, there was aortic occlusion at the renal artery level, with bilateral renal artery stenosis.

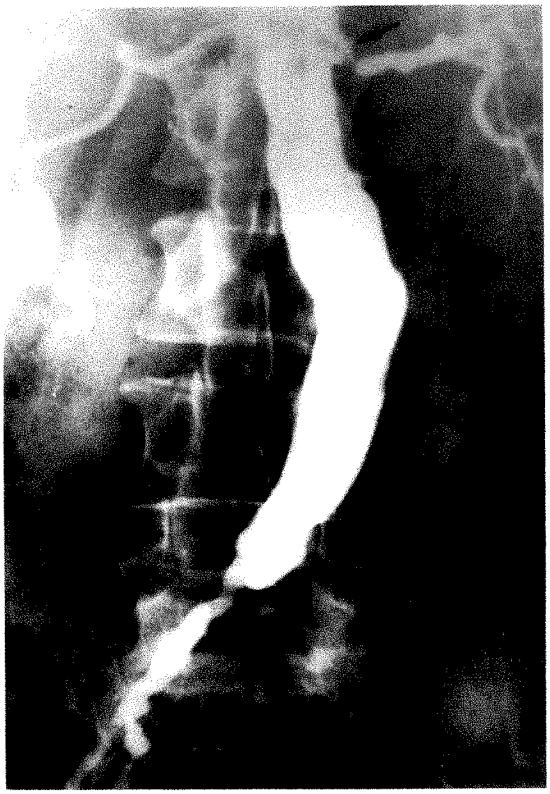

Fig. 9. A 64 year old, severely diabetic man with hypertension and severe bilateral lower limb claudication. The left common iliac artery is occluded and the left renal artery (arrow) is stenosed. The severe atherosclerosis is manifested by the changes apparent in the aorta and right iliac vessels. Note the absence of lumbar arteries due to involvement of their ostia by this process.

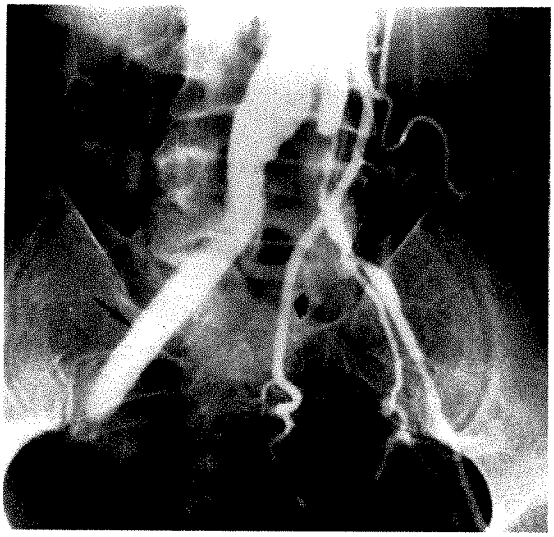

Fig. 10. A percutaneous left axillary femoral arteriogram in a 56 year old man with a long history of peripheral vascular symptoms and previous bilateral aorto-common femoral bypass grafts. The left graft is occluded and note the diffuse stenosis of the left common, internal and external iliac arteries. The right bypass graft (thin arrow) remains patent. The superior hemorrhoidal artery (thick arrow) is dilated and serves as a collateral channel.

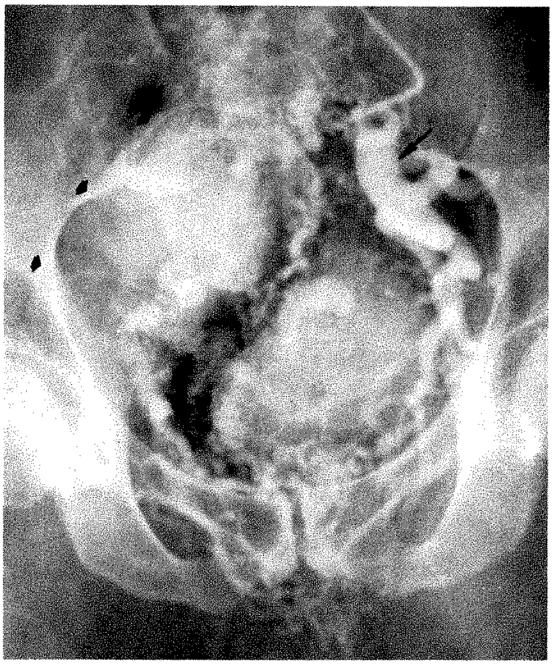

Fig. 11. Selective left internal iliac arteriogram in a 53 year old man with a traumatic aneurysm of the right external iliac artery. The dilated left internal iliac artery (thin arrow) provides innumerable tortuous collateral channels that supply a large calcified vascular chamber (arrow heads) in the right pelvis. The contrast filled bladder is displaced to the left.

of when the red catheter is withdrawn and the guide wire maintained in place. When both the PE240 catheter and guide wire are tip to tip, the PE240 is at the correct level. The difference in length between the catheter and guide wire is measured before introduction of either the wire or catheter into the artery, and this measurement determines when both are tip to tip after insertion. The tip occluder is inserted after the guide wire is withdrawn and the PE240 catheter positioned at the desired level.

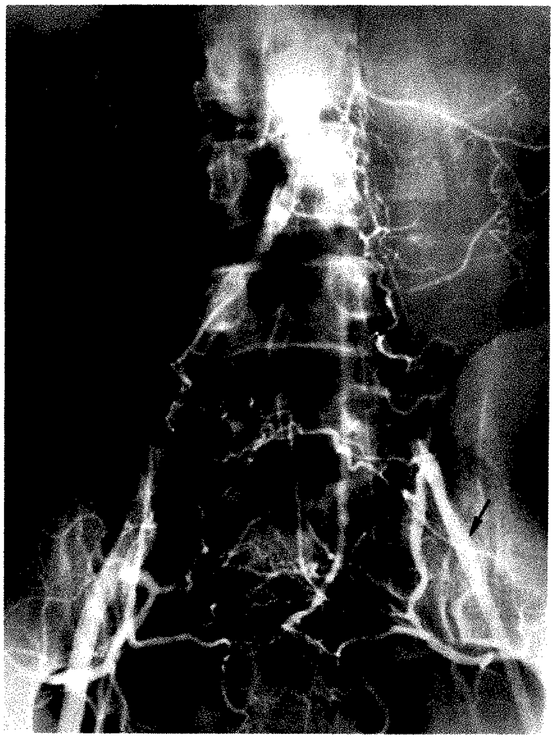

Fig. 12. The late arterial phase of a percutaneous left axillary femoral arteriogram in a 49 year old woman with left leg claudication. The left common iliac artery is occluded. Collateral circulation from the lumbar, middle sacral and presacral areas reconstitutes the left internal and external (arrow) iliac arteries. The right side is normal.

COMMENT

The difficulty of entering the descending aorta from the left axillary artery in the older age group has been pointed out by Bosniak.[1] With age, the aorta elongates, altering the angle between the left subclavian artery and the aorta, causing a catheter to enter the ascending rather than the descending aorta. He concluded that in 100 per cent of patients over the age of 50 and in 50 per cent between 40–50 years, this altered relationship prevailed. Riley *et al.*[6] described a double catheter technique whereby one catheter is advanced through another in order to solve this problem of catheterization. They report only on total aortography by their technique and make no mention of its usefulness for selective visceral arteriography.

We began using the described technique in March, 1964, and since that time have successfully catheterized the abdominal aorta in more than 85 patients by this approach. There have been no failures in this group despite the fact that, in the majority of these patients, the guide wire was initially deflected into the ascending aorta.

COMPLICATIONS

No serious complications have been encountered as a result of this replacement procedure. Initially, the patients complain of tingling in their finger tips and a degree of motor weakness occurs, depending upon the success of the local brachial anesthesia. On follow-up, no cases of permanent motor weakness have been recorded. The frequency and degree of hematoma formation at this site is no different than that encountered from routine percutaneous catheterization elsewhere. No hematoma formation occurs when the PE240 catheter replaces the red Ödman-Ledin catheter, because the former has a larger outer diameter than the latter. So far, we have encountered no cases of acute or gradual thrombosis resulting from this procedure.

SUMMARY

A technique of percutaneous catheterization from the left axillary artery of the abdominal aorta and its branches, in the older age group, is presented. This modification is necessary because of the altered relationship between the left subclavian artery and the descending aorta with advancing age. The ease of performance and lack of serious complications recommend this procedure for investigating thoracic and abdominal aortic lesions when the femoral route is unavailable.

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ABERRANT RIGHT SUBCLAVIAN ARTERY*

CLINICAL AND ROENTGENOLOGIC ASPECTS

By ADRIANUS C. KLINKHAMER
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AN ABERRANT right subclavian artery arises as the last brachiocephalic branch of the aorta. It crosses the mediastinum obliquely from below and the left upward and to the right, reaching the right half of the body (Fig. 1, *A* and *B*).

This paper is based on a series of 50 cases in which an aberrant right subclavian artery was diagnosed roentgenologically. The literature has been reviewed from 1763 to 1962. From 76 publications, 292 necropsy and surgical cases have been collected. Publications in which the origin of the other brachiocephalic branches was not described have been excluded. Three personal observations were verified at surgery and are added.

The purposes of the author is: (1) To show that an aberrant right subclavian artery occasionally is of real clinical significance. It can produce tracheo-esophageal compression in the same way that vascular rings do. (2) To provide an answer to the question of why and when an aberrant right subclavian artery gives rise to clinical manifestations. In our opinion, it is important to emphasize these points, since it was recently stated by Beabout *et al.*[4] that: "The role an aberrant right subclavian artery has in the production of dysphagia in adults or feeding problems and other symptoms in infancy is greatly overrated. It is doubtful that this isolated anomaly ever produces significant dysphagia." (3) To stress some roentgenologic aspects from the diagnostic point of view.

ANATOMIC ASPECTS

In most instances, the aberrant right subclavian artery stems from the dorsal margin of the aortic arch, its origin being between the bend of the top of the arch and the point where the arch lies against the vertebral column.[22,35] In a few cases, the aberrant artery originates from the descending aorta.[22,25,26] The proximal part of the artery is generally greater in diameter than the distal part.[10,22,28,35] In nearly all cases, the aberrant right subclavian artery passes through the mediastinum behind the esophagus; on very rare occasions, it passes between the esophagus and trachea.[3,5] Publications describing a pretracheal course of the aberrant vessel are not very convincing.[4,25]

In 85 of the 295 cases (29 per cent) collected from the literature and from personal observation, the aberrant right subclavian artery was found to be associated with a common origin of the right and left carotid arteries, the bicarotid truncus. In 31 cases (10 per cent), the 2 carotid arteries were situated closer to each other than normally, although they were still separated. In 179 cases (60 per cent), a normal origin of the carotid arteries was reported. In our opinion, it is this abnormal origin of the carotid arteries, combined with the aberrant right subclavian artery, that provides an explanation for the clinical manifestations produced by this aberrant vessel (see clinical aspects).

CLINICAL ASPECTS

The term "dysphagia lusoria" or difficulty in swallowing due to a trick of nature (aberrant artery) was introduced by Bayford[3] in 1787. Hamburger[20] and Hyrtl[24] were among the first to deny a relationship between dysphagia and an aberrant right subclavian artery. Hamburger proposed to change the term of dysphagia lusoria to "dysphagia illusoria." In the literature of more recent date, however, dysphagia due

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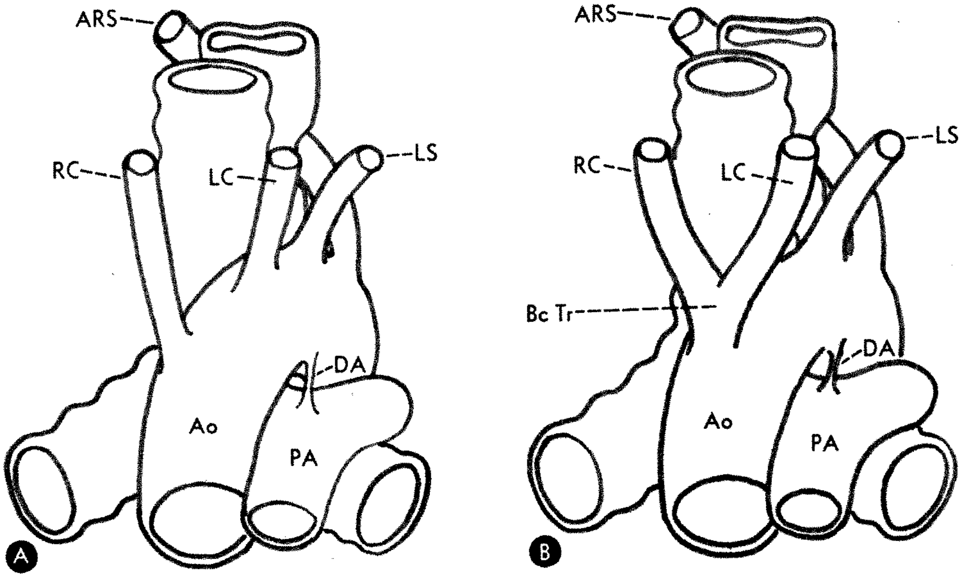

Fig. 1. Diagrammatic representation of the aberrant right subclavian artery. (*A*) Normal origin of the carotid arteries. Trachea and esophagus can shift forward by using the space between the two carotid arteries. (*B*) Common origin of the carotid arteries: bicarotid truncus. The V in front of the trachea prevents the trachea and esophagus from bending forward. Both organs are hemmed in, dorsally by the aberrant right subclavian artery and ventrally by the carotid fork. Ao = aorta; PA = pulmonary artery; DA = ductus arteriosus; RC = right carotid artery; LC = left carotid artery; LS = left subclavian artery; ARS = aberrant right subclavian artery; BcTr = bicarotid truncus.

to the aberrant vessel has been reported frequently.[2,9,14,15,17,25,26,29,33,37] On the other hand, Beabout *et al.*[4] stated that "it is doubtful that this isolated anomaly ever produces significant dysphagia." Of great importance, although less widely known, is the fact that, particularly in childhood, an aberrant right subclavian artery can cause serious respiratory symptoms, without the presence of dysphagia. In children, tracheo-esophageal compression manifests itself by respiratory symptoms. In adults, the trachea is more rigid and, therefore, more resistant to compression. Here dysphagia is the principal complaint.[8,21,28] This applies to both, compression produced by vascular rings and that produced by an aberrant right subclavian artery.

In 9 of our 50 cases, signs of tracheo-esophageal compression were found. Two children had serious respiratory symptoms, necessitating surgical intervention in one child. The remaining 7 patients, all adults, had a history of dysphagia.

Ever since the nineteenth century, one repeatedly encounters the question why and when do these anomalies give rise to clinical manifestations.[12,22,27,31] An artery pressing on the esophagus from behind is not in itself a sufficient explanation for the occurrence of compression. Patients having very large arteries retro-esophageally, as found in some types of right-sided aortic arch, have been noted to have no respiratory distress or dysphagia. It is our belief that these symptoms arise only if the flexible trachea and esophagus are hindered from being bent forward at the crossing with the retro-esophageal vessel. This can occur when the right and left carotid arteries arise together or close to each other from the aortic arch (Fig. 1, *A* and *B*).

Normally, the innominate artery and the left carotid artery originate from the arch at a distance of about 4 cm. from each other. The arch lies obliquely in the chest and curves from the front and the right backward and to the left. Accordingly, the proximal part of the innominate artery lies more ventrally than the proximal part of the left carotid artery. If an aberrant right subclavian artery is present, the trachea and the esophagus can escape compression, exerted by the retro-esophageal vessel being bent forward. In this situation, the space between the 2 carotid arteries is utilized (Fig. 1*A*). If the right and left carotid arteries arise together or in close proximity (Fig. 1*B*), then their proximal parts lie in the same frontal plane, forming a V configuration in front of the trachea. It is this fork which prevents the trachea and the esophagus from being bent forward. The esophagus and the trachea are hemmed in, dorsally by the aberrant right subclavian artery and ventrally by the 2 carotid arteries. The result is not only impression, but also compression of both organs. The case below is an illustrative example of such an instance.

M.v.M., No. 90190. One month after birth on September 16, 1956, this girl was hospitalized because of choking spells and a stridor that could be heard from a distance. During 4 months of hospitalization, the child suffered from frequent relapsing infections of the respiratory tract. No cause could be found. She was re-admitted 4 times thereafter because of persistent stridor, recurrent lung infections and lack of growth. Not until 2 years after the first admission were studies of the esophagus done. The esophagrams showed the typical picture of an aberrant right subclavian artery. An operation revealed a normal aortic arch. The most proximal branch was a short, thick stem, which split into 2 branches—the 2 carotid arteries. They formed a V configuration in front of the trachea. The right subclavian artery passed behind the esophagus. The trachea and the esophagus were fixed between these arteries.

Our conception about the cause of compression is not based on this single observation alone. From 76 publications we collected 292 cases found in the literature. A bicarotid truncus or a close origin of the carotid arteries was found in 116 cases (39 per cent). Only in these cases were symptoms and signs of tracheo-esophageal compression noted.

We feel that the combination of an aberrant right subclavian artery and an abnormal origin of the carotid arteries is a *conditio sine qua non* for the occurrence of compression. Undoubtedly, many of these cases will not produce clinical manifestations. This is due to the fact that compression only occurs if: (1) the common or close origin of the carotid arteries is such that anterior evasion of the trachea is prevented, and (2) the space around the trachea and esophagus is narrow enough to prevent an escape from compression in any direction.

Bailey,[2] Ekström,[9] and Gross[15,19] believe that the respiratory distress is caused by aspiration as a consequence of the dysphagia. In our opinion, respiratory distress occurs because the trachea is also enclosed and compressed. This is supported by several observations of Bailey and Gross. Thus, Gross[16] describes the histories of 9 young patients with serious respiratory distress. At operation, the innominate artery was found to arise from the arch too far to the left and to compress the trachea. By lifting the artery from the trachea, dramatic improvement resulted. Five other patients with the same complaint were found to have the left carotid artery arising from the arch farther to the right than normally. In these cases also, fixation of the artery to the sternum led to good results.

Bailey reported a case in which the innominate artery and the left carotid artery originated together from a large truncus. The carotid arteries passed upward on each side of the trachea, which was wedged and compressed between the two arteries.

Obviously, if variations in the origin of the carotid arteries can lead to compression of the trachea on their own, this compression is enhanced if, along with it, the esophagus is impressed from behind by a retro-esophageal right subclavian artery.

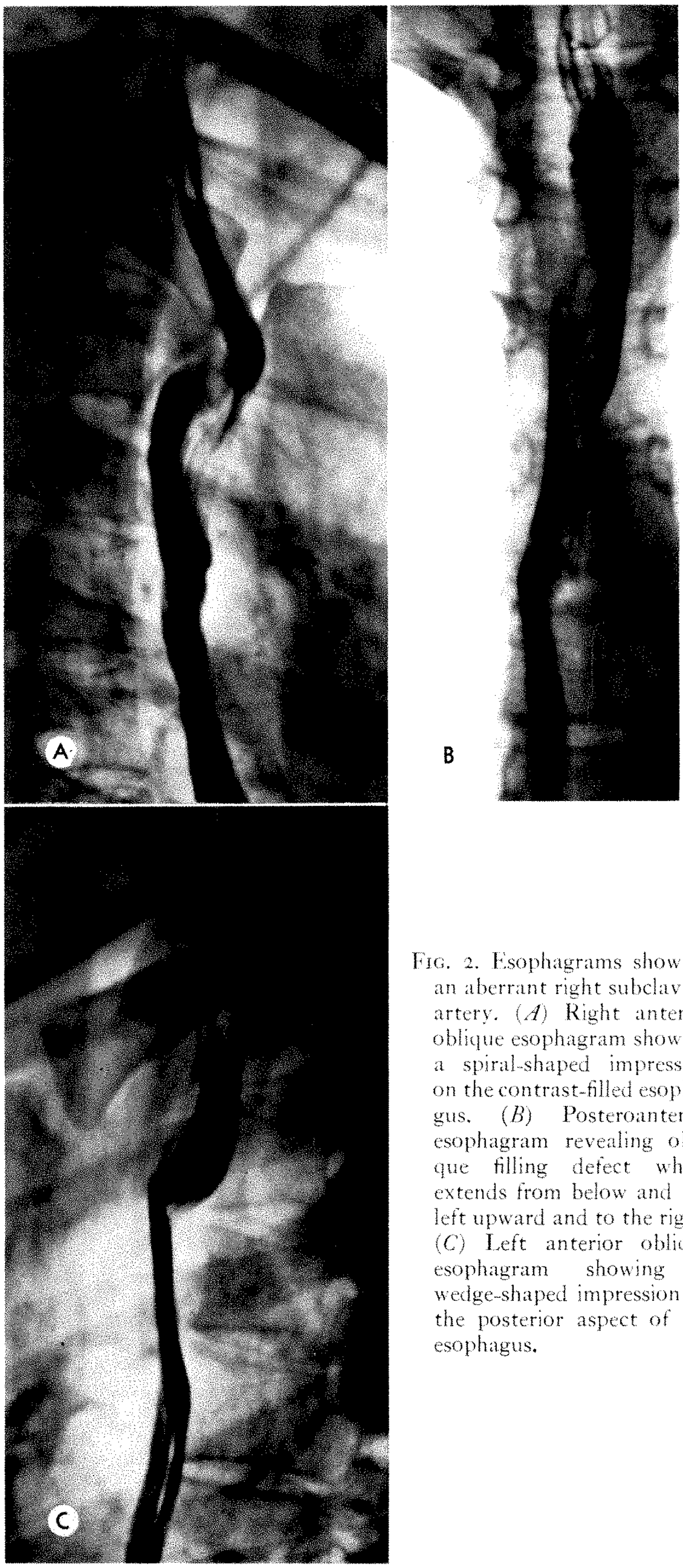

FIG. 2. Esophagrams showing an aberrant right subclavian artery. (*A*) Right anterior oblique esophagram showing a spiral-shaped impression on the contrast-filled esophagus. (*B*) Posteroanterior esophagram revealing oblique filling defect which extends from below and the left upward and to the right. (*C*) Left anterior oblique esophagram showing a wedge-shaped impression on the posterior aspect of the esophagus.

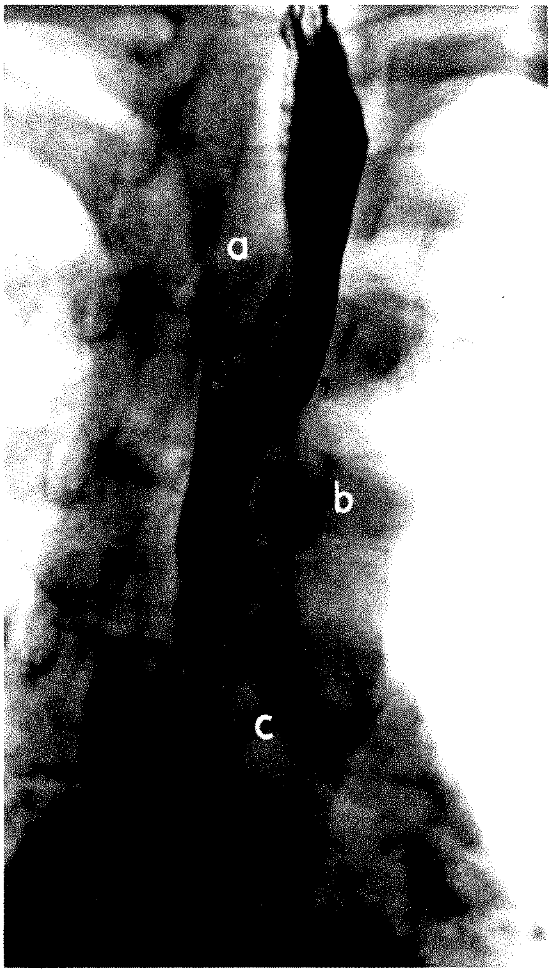

FIG. 3. Posteroanterior esophagram showing indentations produced by the aberrant right subclavian artery (a), the aortic arch (b), and the left main bronchus (c).

ROENTGENOLOGIC ASPECTS

The posteroanterior esophagram (Fig. 2*B*) demonstrates, at the level of the aortic arch or just above it, an oblique filling defect in the contrast-filled esophagus, extending from below and the left upward and to the right.[6,11,12,32] The parts of the esophagus lying orally and aborally to the defect are simultaneously displaced in the sense of a *dislocatio ad latum* in a bone fracture ("fracture avec chevanchement,"[5] "deviazione a baionetta"[30]). In the right anterior oblique esophagram (Fig. 2*A*), the esophagus is seen to turn upward, like a spiral, just above the aortic arch. In the left anterior oblique projection (Fig. 2*C*) and in the lateral view, a wedge-shaped impression on the dorsal aspect of the contrast-filled esophagus may be noted.

The left anterior oblique projection of the esophagus is best for differentiating between an indentation due to an aberrant right subclavian artery and an indentation produced by the left main bronchus. This applies particularly in young children, in whom the aortic arch cannot always be visualized.

In some instances, the impressions of the left main bronchus, the aortic arch and the aberrant artery can be distinguished very clearly (Fig. 3). However, the origin of the aberrant artery can lie more caudally (see anatomic aspects) and in these cases, the indentation of the aberrant vessel in the posteroanterior esophagram is seen on level with the bifurcation of the trachea. If the patient is placed in the right anterior oblique position, the left main bronchus and the aberrant artery are projected as extensions of each other. An esophagram in this position shows the translucency of the left main bronchus terminating in an indentation in the esophagus (Fig. 4*A*). In the left anterior oblique position, the left main bronchus and the aberrant vessel are well separated, allowing clear identification of the wedge-shaped indentation due to an aberrant right subclavian artery lying higher than the translucency produced by the left main bronchus (Fig. 4*B*).

In 12 of the 47 cases in which esophagrams were made in both oblique views, the left anterior oblique esophagram allowed the conclusion that the impression was caused by an aberrant right subclavian artery.

In elderly patients, the oblique esophagrams do not show convincingly the presence of the aberrant right subclavian artery; the marked indentations of the aortic arch on the barium-filled esophagus mask the characteristic picture (Fig. 5, *A*, *B* and *C*). The posteroanterior esophagram may prove of greater value. In this view, there is a definite indentation at the level of the crossing (aberrant vessel and esophagus).

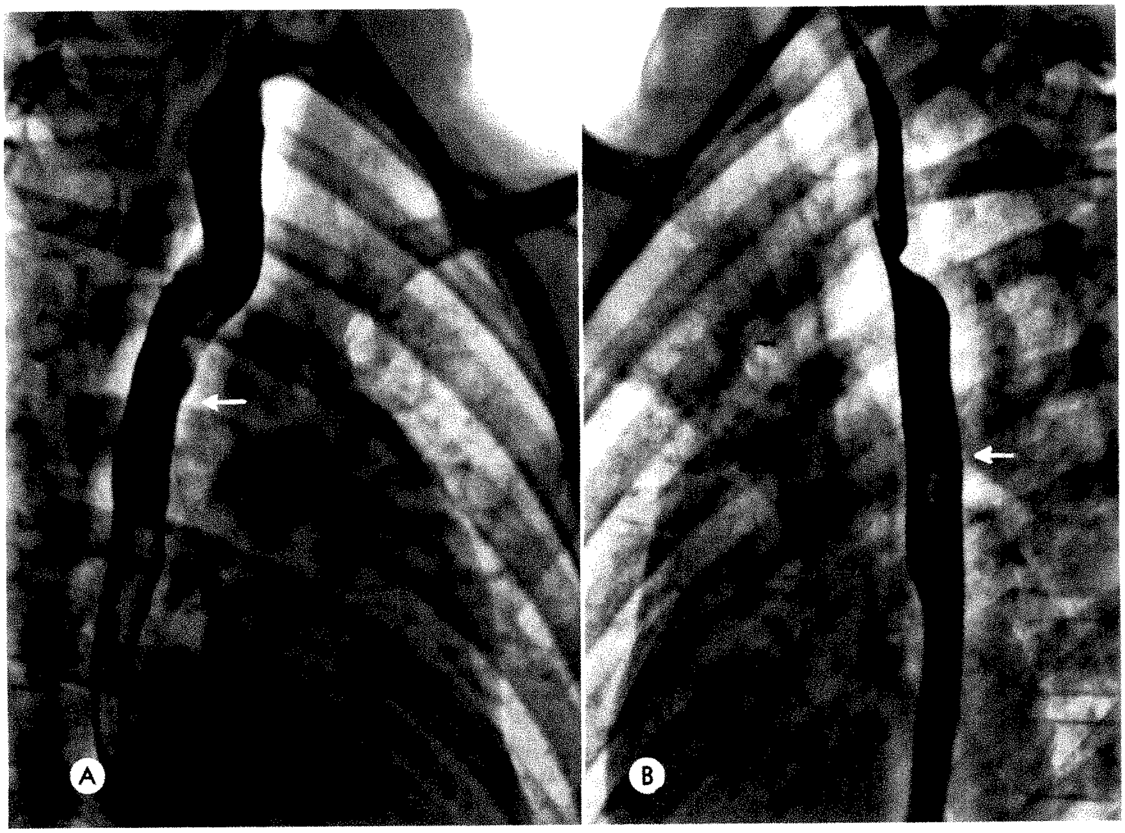

Fig. 4. Differentiating points between an impression due to an aberrant right subclavian artery and one produced by the left main bronchus. (*A*) Right anterior oblique esophagram. The translucency of the left main bronchus ends in a spiral-shaped impression in the esophageal wall. Impression (←) caused by an aberrant artery or by the left main bronchus? (*B*) Left anterior oblique esophagram. Now it becomes clear that the indentation is produced by the aberrant right subclavian artery. The translucency of the left main bronchus (←) is situated far more inferiorly.

Between this indentation and the shadow of the aortic arch, a curved space may be visible, identifying the aberrant artery.

Figure 6, a lateral esophagram of the 2 year old patient here reported, shows tracheo-esophageal compression produced by the aberrant artery. In the posterior margin of the esophagus, a wedge-shaped indentation is visible. At the level of this indentation, the esophagus does not bend forward but continues downward in a straight line. The trachea lies close to the anterior wall of the esophagus and, likewise, does not bend forward at the indentation in the dorsal margin of the esophagus. The anterior border of the trachea is flattened by the fork formed by the carotid arteries. This was confirmed at operation.

SUMMARY

An aberrant right subclavian artery can occasionally give rise to clinical manifestations. Dysphagia as a symptom has long been known; however, in children, serious respiratory distress may occur, even in the absence of dysphagia.

The retro-esophageal course of the right subclavian artery does not in itself account satisfactorily for the tracheo-esophageal compression, which can only occur if the carotid arteries present a common origin from the aortic arch or if they are in close approximation. This belief is based on a personal observation as well as a review of 292 necropsy and surgical cases, collected from 76 publications in the literature.

The diagnosis was made from the esopha-

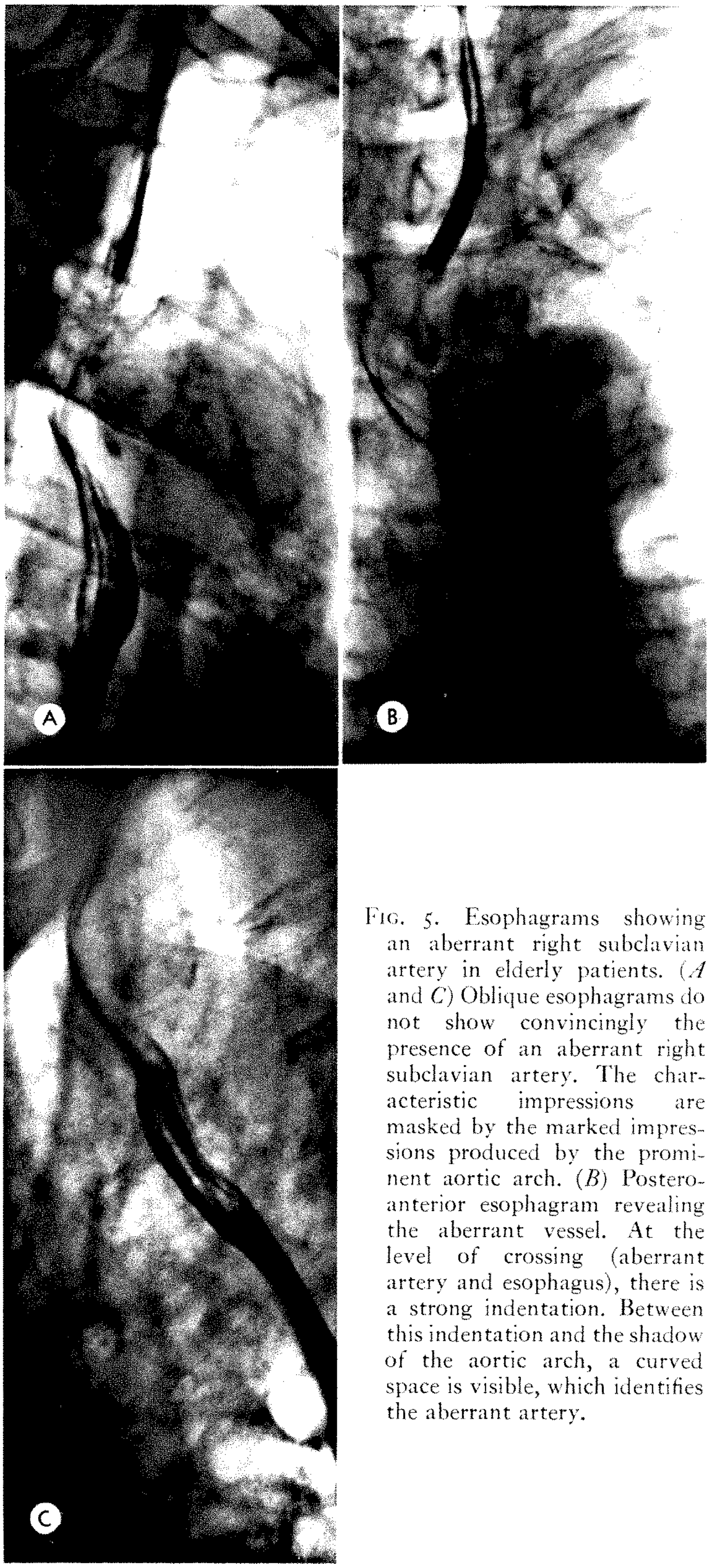

FIG. 5. Esophagrams showing an aberrant right subclavian artery in elderly patients. (*A* and *C*) Oblique esophagrams do not show convincingly the presence of an aberrant right subclavian artery. The characteristic impressions are masked by the marked impressions produced by the prominent aortic arch. (*B*) Posteroanterior esophagram revealing the aberrant vessel. At the level of crossing (aberrant artery and esophagus), there is a strong indentation. Between this indentation and the shadow of the aortic arch, a curved space is visible, which identifies the aberrant artery.

grams in 50 cases. Clinical signs of tracheo-esophageal compression were found in 9. Two children presented with respiratory distress, necessitating surgical intervention in one of them. Dysphagia was noted in 7 cases, all adults.

The indentations in the barium-filled esophagus caused by the aberrant vessel are discussed. Attention is drawn to the importance of the left anterior oblique esophagram for distinguishing a filling defect caused by an aberrant right subclavian artery from one produced by the left main bronchus. In elderly patients, the postero-anterior esophagram is of greater value in establishing the diagnosis.

The roentgenologic characteristics of tracheo-esophageal compression produced by the aberrant vessel are demonstrated in the lateral esophagram (Fig. 6) of a 2 year old girl who was operated upon because of serious respiratory distress.

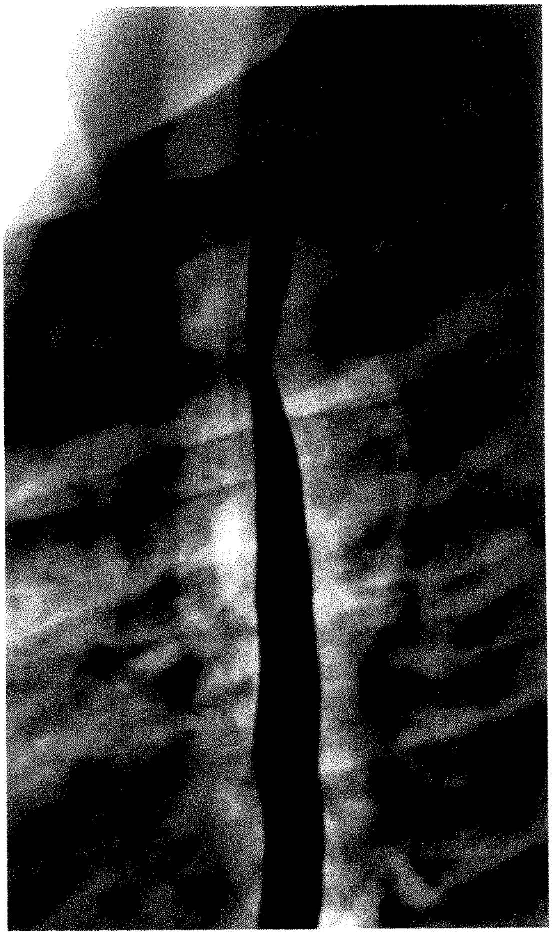

Fig. 6. Two year old girl with serious respiratory distress. Operation disclosed an aberrant right subclavian artery and bicarotid truncus. Lateral esophagram showing a wedge-shaped indentation on the posterior aspect of the esophagus. At this impression, the esophagus shows no anterior displacement. The anterior wall of the trachea is flattened by the carotid arteries, as found at operation.

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TWO UNUSUAL CASES OF SUBCLAVIAN STEAL SYNDROME*

BILATERAL STEAL AND STEAL SECONDARY TO TUMOR THROMBUS

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IN A 1961 *New England Journal of Medicine* editorial,[4] there was coined the term "subclavian steal" to describe the condition where circulation to an arm is maintained around a proximal subclavian artery occlusion by way of collateral channels (usually vertebral), thus "stealing" blood which normally might have supplied intracranial structures. Since then, numerous cases have been reported and most of the usual as well as the less common routes of collateral flow postulated by anatomists have been substantiated clinically.[1,2,10,11]

The cases reported here are unusual examples of this syndrome. One case, possibly an example of Takayasu's syndrome as well, is unusual in the multiplicity of obstructions and pathways of collateral flow, while the other, caused by partial obstruction of the subclavian artery by tumor thrombus, represents a rare and heretofore unreported cause for the steal syndrome.

REPORT OF CASES

CASE I. A 65 year old retired female school teacher who had smoked heavily for 30 years gave a history of 5 months of impaired vision and pain in the right eye which had increased in severity over the 6 week period preceding admission. The visual disturbances were accompanied by lacrimation, swelling of the right periorbital tissues, diplopia, and blurred vision, although the latter had been largely corrected by modification of her spectacles. On the basis of a diagnosis of pseudotumor of the orbit, steroid therapy was instituted, followed by some relief of symptoms. Quite possibly, however, the visual difficulties were partly a result of the severe vascular disease presently to be described.

The patient also recalled that for several years her arms had tired quickly when they were raised above the horizontal, and that occasionally her fingers tingled or were numb. She remembered too that for 40 years physicians had told her that the blood pressure in her arms was low (80 mm. Hg systolic). The family history was not significant.

On physical examination, there were found slight right sided proptosis and periorbital edema. Presystolic bruits could be heard over the posterior part of the neck from the occiput to C7, in both supraclavicular areas, over both orbits (particularly left), the abdominal aorta, and over both femoral areas. In the arms blood pressure was 80/60, in the legs 250/70. There was minimal swelling in the interphalangeal joints thought to represent rheumatoid arthritis; reflexes were normal.

Serum cholesterol levels, serologic tests for syphilis, multiple lupus erythematosus preparations and rheumatoid agglutinins were normal. Corrected sedimentation rates averaged 10 mm., and serum protein was 5.9 gm. per cent with a normal electrophoretic distribution. A right temporal artery biopsy was normal.

Skull roentgenograms, including orbital views, were normal. Arch aortography was performed, with right femoral catheter approach, and bilateral subclavian obstruction was found, with poor filling of both carotid arteries (not shown). No internal mammary or intercostal collaterals were present. Subsequently, bilateral percutaneous carotid arteriograms were made (Fig. 1 through 4, inclusive; routes of blood flow are shown better in the accompanying drawings than in reproductions of the roentgenograms themselves, so only the initial roentgenogram of each series is included). Space prohibits inclusion of all of the pertinent roentgenograms, but on those showing the intracranial circulation, it was apparent that the left external carotid through its ophthalmic and vertebral anastomoses carried the bulk of blood to both sides of the brain. The small right ophthalmic and right internal carotid arteries

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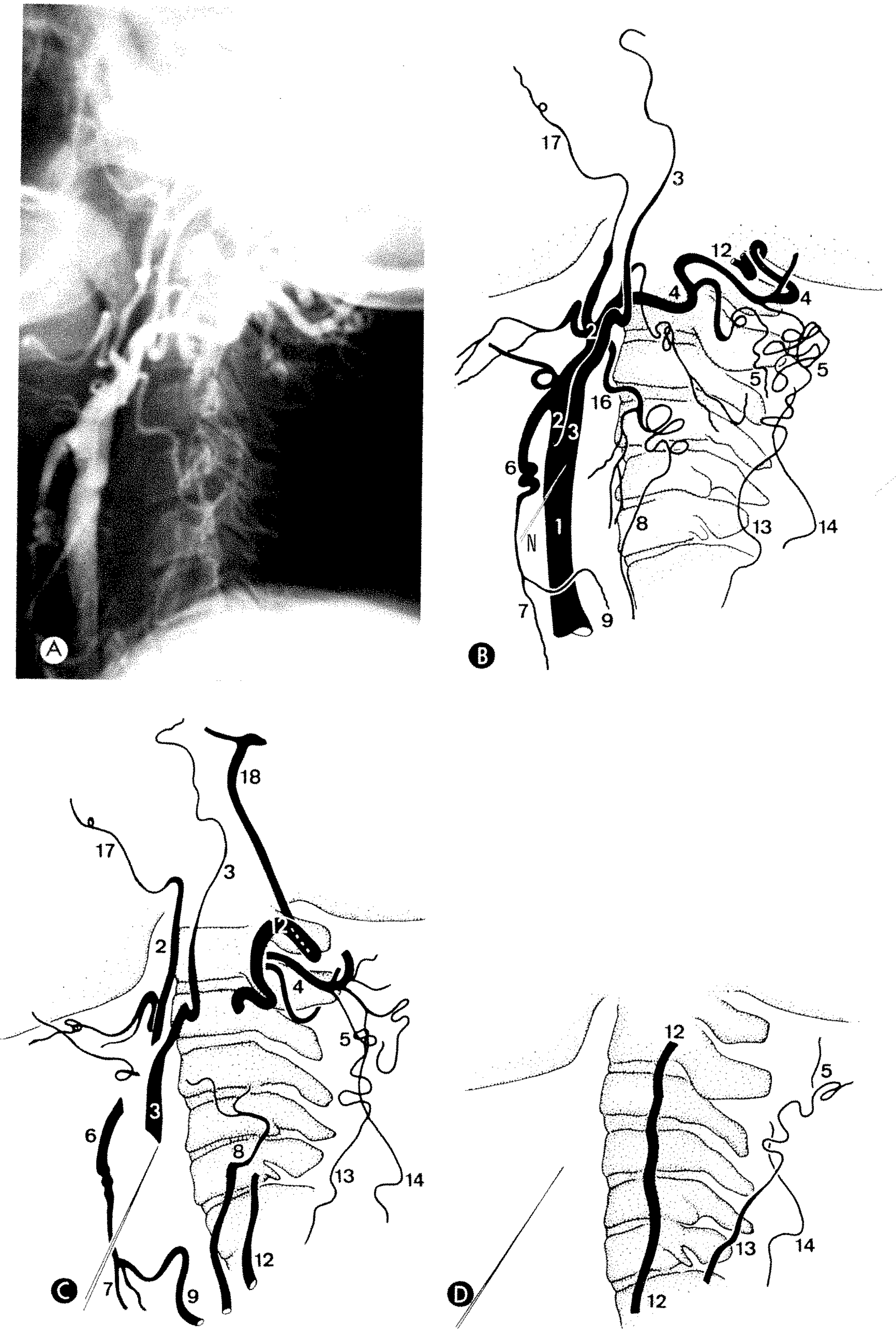
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carried little blood, and the right vertebral artery contributed a lesser amount.

Surgical correction was deemed impractical. The patient was discharged with instructions to take analgesics and discontinue smoking, and periodically has been retreated with steroids.

Comment. In this one patient, therefore, there are utilized virtually all of the possible collateral routes in the neck for proximal subclavian obstruction. The abnormal carotid circulation explains the failure of the circle of Willis to furnish a substantial collateral route for downward flow into the arms and bilaterality explains nonutilization of the internal mammary arteries; but the failure to use the intercostal arteries cannot be explained. Undoubtedly, the longstanding nature of this patient's occlusive disease, allowing time for development of adequate collaterals, accounts for her rather striking lack of symptoms. In spite of the atypically long clinical course and site of some of the areas of occlusion (carotid arteries), it seems probable that this is a case of Takayasu's syndrome, and the otherwise inexplicable uniform diminution in caliber of the right internal carotid artery, a generalized smooth narrowing, is compatible with previous reports of the roentgenographic features of that disease.[8] To the best of my knowledge, this is the fifth case of the bilateral steal syndrome to be roentgenographically documented,[2,3,8,10] and the second case of Takayasu's syndrome with accompanying steal.[8] (There are several other cases in the literature, some reported before identification of the subclavian steal as an entity, which might be other examples of a "double steal," but they were either not recognized as such or

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FIG. 1. (*A*) Right carotid arteriogram, lateral view. For details see drawing in *B*.

(*B*) One second following onset of injection of contrast material through a needle (N). The right common carotid artery (1), arising from the innominate artery proximal to the point of obstruction, receives blood in the normal manner. Its internal branch (3) is of abnormally small caliber. The external carotid artery (2) is enlarged because it must supply an abnormal amount of blood to the internal maxillary artery (17), superior thyroidal artery (6), the prevertebral branch of the ascending pharyngeal artery (16), and to the descending cervical and muscular branches (5) of the occipital artery (4). There is faint opacification of the right inferior thyroidal (7), left inferior thyroidal (9), ascending cervical branch of the right inferior thyroidal (8), right deep cervical (13), ascending cervical branch of the transverse cervical (14), and the right vertebral (12) arteries.

(*C*) One second later than *B*. Blood delivered from the occipital artery into the vertebral artery flows bidirectionally in that vessel. The upward flow through the cranial portion of the vertebral artery (12) has filled the basilar artery (18) and its intracranial branches, but the downward flow as yet has progressed only to approximately C3. The lower cervical anastomotic vessels (7, 8, 9, 13, 14) are better seen, being filled with blood flowing in reversed direction from above downward. Another segment of the vertebral artery (12) at approximately C6 has filled.

(*D*) One second later than *C*. Almost the entire vertebral artery (12) has filled by flow retrograde from above. (Its communication with the subclavian artery is shown in Figure 2*D*.)

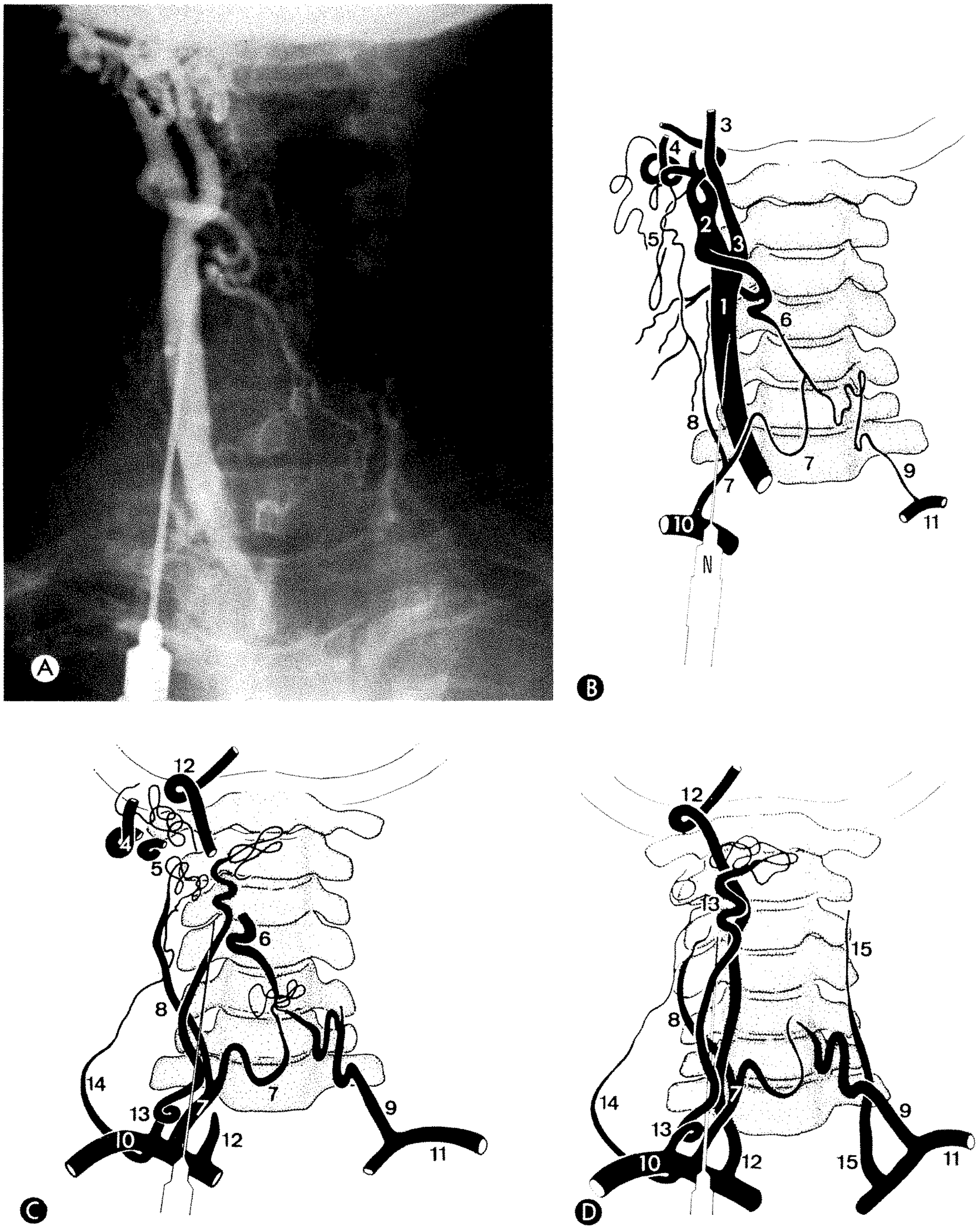

Fig. 2. (*A*) Right carotid arteriogram, frontal view. For details see drawing in *B*.

(*B*) Approximately the same timing as in Figure 1*B*. Common carotid (1), external carotid (2), internal carotid (3), occipital (4), descending and muscular branches of the occipital (5), and superior thyroidal (6) arteries have filled in normal fashion. The ascending cervical branch of the right inferior thyroidal artery (8), both inferior thyroidal arteries (7, 9), and both subclavian arteries (10, 11) (distal to the two points of obstruction) now are receiving blood from above.

(*C*) Approximately same timing as in Figure 1*C*. Blood from the descending cervical and muscular had insufficient roentgenographic or hemodynamic flow pattern explanation to be included.)[5,6,7,9,11,13]

Case II. A 46 year old business man and former aviator experienced 3 transient episodes of left eye blindness and occasional slurring of speech with inability to perform simple computations during the month before admission. The patient aptly described his cerebral hypoxia as similar to the experience of "flying at a high altitude."

Neurologic examination was normal except for markedly diminished pulsation of the left carotid artery and diminished blood pressure in the left arm (left 120/90—right 152/92).

Skull and chest roentgenograms were normal. Right retrograde brachial arteriograms (Fig. 5, *A*, *B* and *C*) demonstrated a left subclavian steal syndrome. Subsequently, an arch aortogram (right femoral catheter approach) was made, showing partial occlusion of the left subclavian artery and complete occlusion of the left carotid artery at its orifice (not shown). The catheter was then positioned with its tip in the left subclavian artery, and two additional series of roentgenograms were made (Fig. 6, *A* and *B*).

At surgery 6 days later, the left common carotid artery was found to be a fibrous cord, unsuitable for repair or bypass, and the partially obstructed portion of the left subclavian artery was resected and replaced by a teflon graft. Pathologic examination of the vessel showed the block to consist of organizing thrombus containing tumor cells of undetermined origin.

Exhaustive investigation failed to show the source of neoplasm. The patient was discharged in a relatively asymptomatic state, his graft functioning well.

Three months later a destructive lesion developed in the mandible, which on biopsy proved to be an undifferentiated neoplasm, pos-

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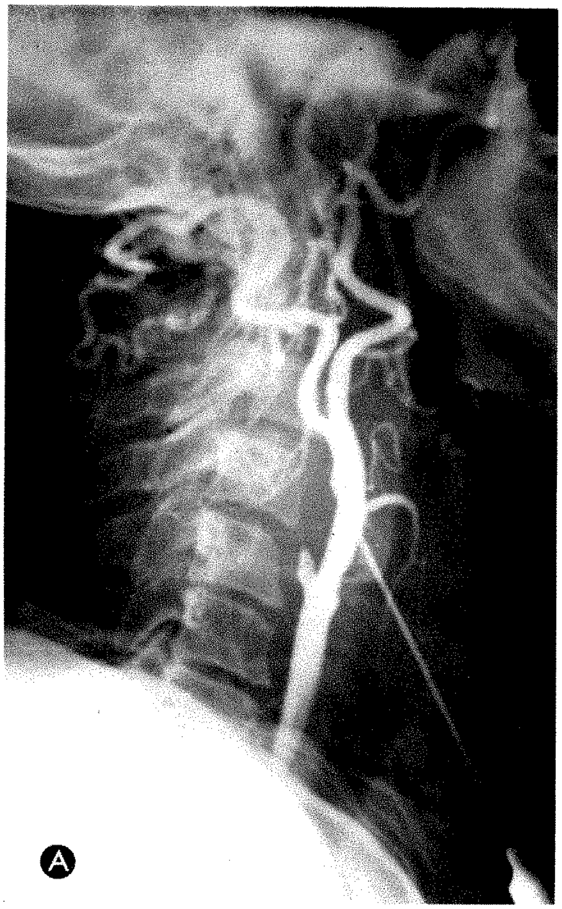
branches of the occipital artery (5) now has better filled the ascending cervical branches of the inferior thyroidal artery (8) and of the transverse cervical artery (14) along with the deep cervical artery (13). The subclavian arteries (10, 11) are seen better than before. As in Figure 1*C*, the upper and lower portions of the vertebral artery (12) are opacified.

(*D*) Approximately the same timing as in Figure 1*D*. The left vertebral artery (15) has filled from above, and as in Figure 1*D*, the right vertebral artery (12) is filled throughout.



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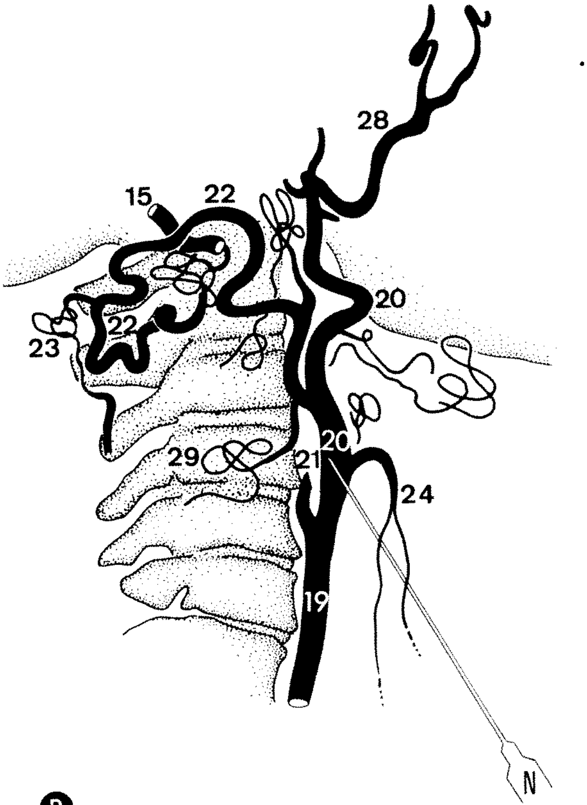
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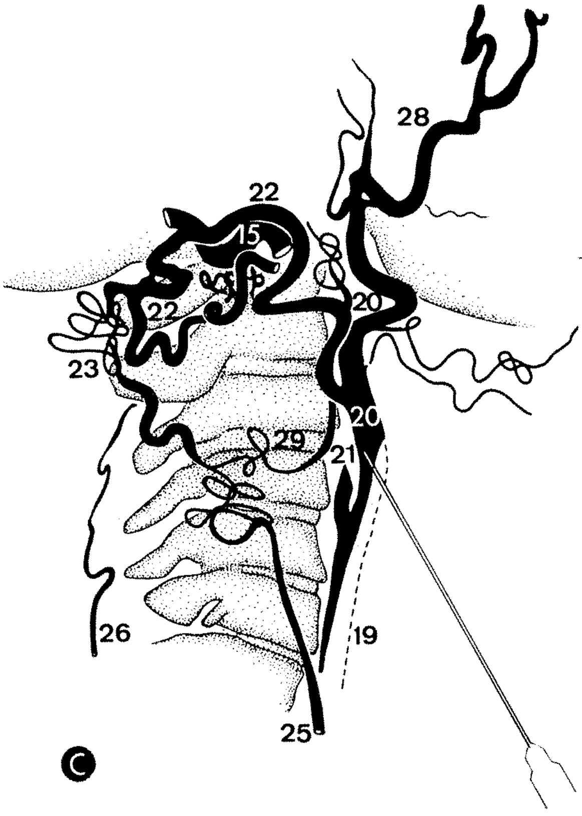
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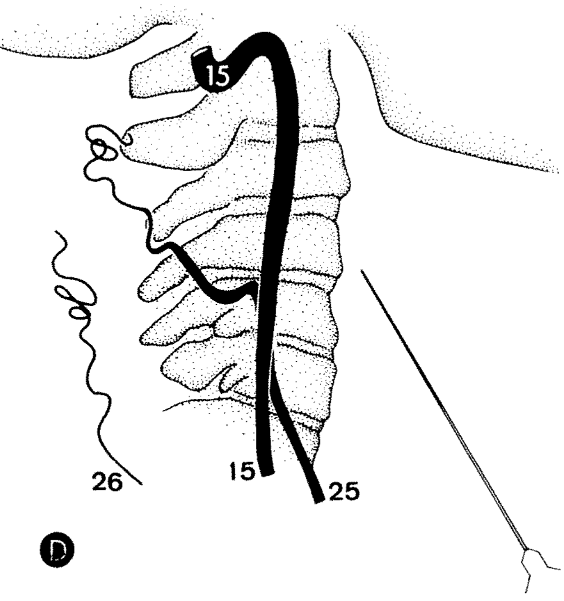
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sibly of renal origin or from an undetected sarcoma. Subsequently, metastases developed in the spine, and like those in the mandible, responded to irradiation.

Seven and one-half months after his initial hospitalization, the patient died and, at autopsy, was found to have widespread metastases in bone, brain, and both kidneys (particularly the right). There was involvement of the chest wall and ribs about the left shoulder and intraluminal tumor had re-occluded the left subclavian artery and was present as well in the left carotid artery, right renal artery and the thoracic aorta. The tumor tissue was poorly differentiated and appeared to have originated in the right adrenal cortex. Whether the peculiar "squared off" sharply localized nature of the obstruction (Fig. 6 *B*) is a useful criterion suggesting intraluminal tumor is not known.

Comment. This case is remarkable in two respects. Other examples of obstruction resulting from compression of vessels by adjacent neoplasm (for example, in superior sulcus pulmonary neoplasm) might be anticipated, but subclavian steal resulting from occlusion by intraluminal neoplasm probably will remain a rarity. The second unusual feature (based on video tape recordings made with a catheter in several different positions within the left subclavian artery during the course of fluoroscopy) is the probability that in spite of a high degree of mechanical obstruction the "steal" is inconstant, or at least incipient. These observations support published reports on the degree of hemodynamic abnormality required to alter the gradient of

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Fig. 3. (*A*) Left carotid arteriogram, lateral view. For details see drawing in *B*.

(*B*) One second following onset of injection of contrast material. Contrast material injected into the external carotid artery (20) has filled the superior thyroidal artery (24), internal maxillary artery (28), prevertebral branch of the left ascending pharyngeal artery (29), the occipital (22), and its descending cervical and muscular branches (23), and in addition, has refluxed against arterial pressure to fill the common carotid artery (19) and the stump of its occluded internal branch (21). There is minimal filling of the left vertebral artery (15) from its muscular branch anastomoses with occipital branches.

(*C*) One second later than *B*. The vertebral artery (15) is better filled. The downreaching anastomoses of the descending cervical and muscular branches (23) of the occipital (22) and the prevertebral branches of the ascending pharyngeal artery (29) are delivering opacified blood to the ascending cervical branches of the left inferior thyroidal (25) and the transverse cervical (26) arteries. (Not shown in these figures is blood flow from the left internal maxillary artery (28) through the large left ophthalmic artery and thence retrograde into the carotid siphon.)

(*D*) One second later than *C*. The entire left vertebral artery (15) is filled by retrograde flow.

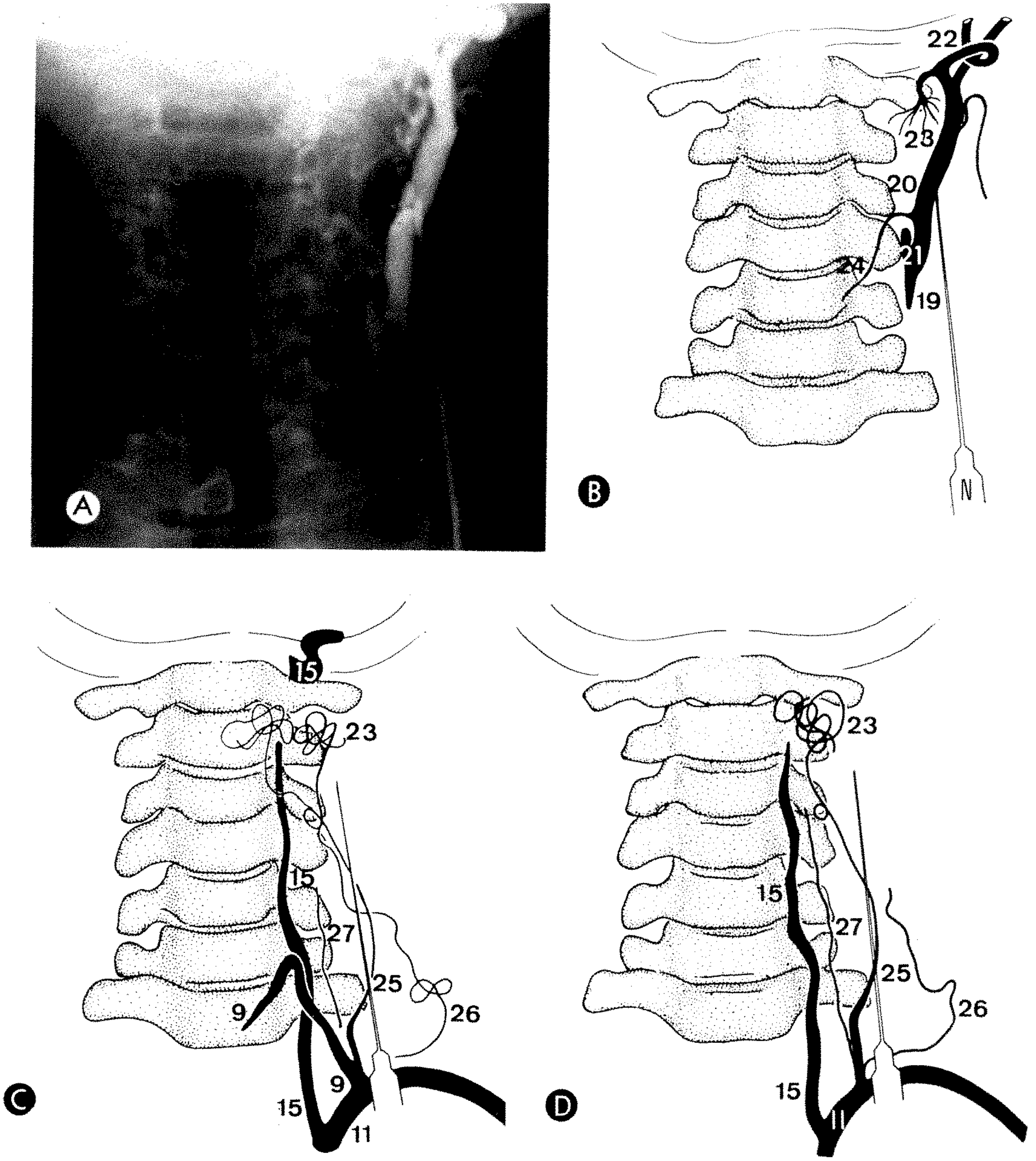

Fig. 4. (*A*) Left carotid arteriogram, frontal view. For details see drawing in *B*.

(*B*) Approximately same timing as in Figure 3*B*. As in Figure 3*B* contrast material injected into the external carotid artery (20) has filled the occipital (22) and the superior thyroidal (24) arteries and has refluxed into the common carotid artery (19) and the stump of its internal branch (21).

(*C*) Approximately same timing as in Figure 3*C*. The entire left vertebral artery (15) has filled from above. (Some segments not well seen.) As on the right side, anastomoses have been established over the deep cervical artery (27), and ascending cervical branches (25) of the inferior thyroidal (9) and of the transverse cervical arteries (26), all receiving blood from the muscular and descending cervical arteries (23). The inferior thyroidal artery (9) brings blood down from the superior thyroidal artery as well. The left subclavian artery (11) is beginning to fill.

(*D*) Approximately same timing as in Figure 3*D*. The distal portion of the subclavian artery (11) and the inferior portion of the left vertebral artery (15) show better than in *C*.

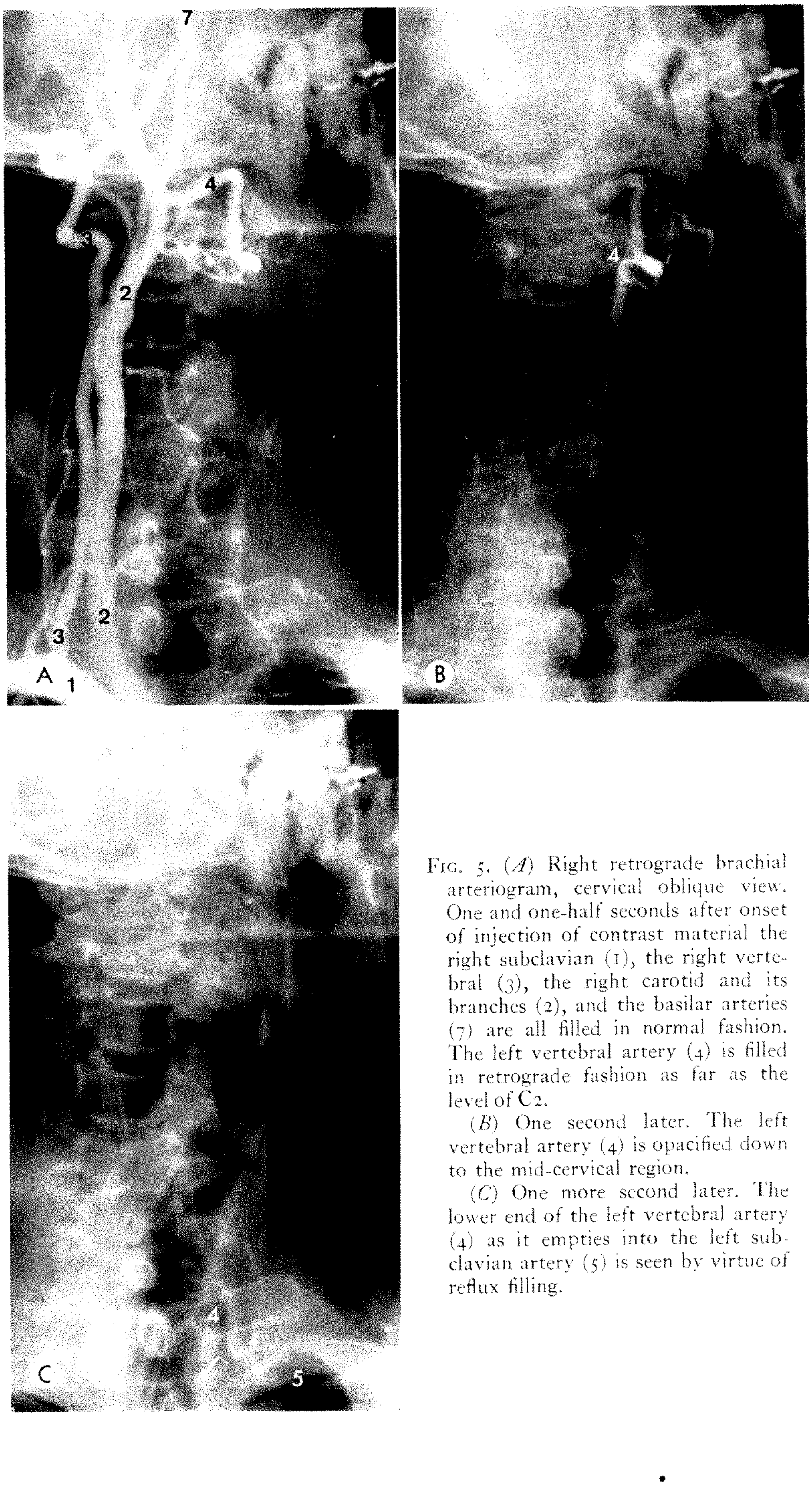

Fig. 5. (*A*) Right retrograde brachial arteriogram, cervical oblique view. One and one-half seconds after onset of injection of contrast material the right subclavian (1), the right vertebral (3), the right carotid and its branches (2), and the basilar arteries (7) are all filled in normal fashion. The left vertebral artery (4) is filled in retrograde fashion as far as the level of C2.

(*B*) One second later. The left vertebral artery (4) is opacified down to the mid-cervical region.

(*C*) One more second later. The lower end of the left vertebral artery (4) as it empties into the left subclavian artery (5) is seen by virtue of reflux filling.

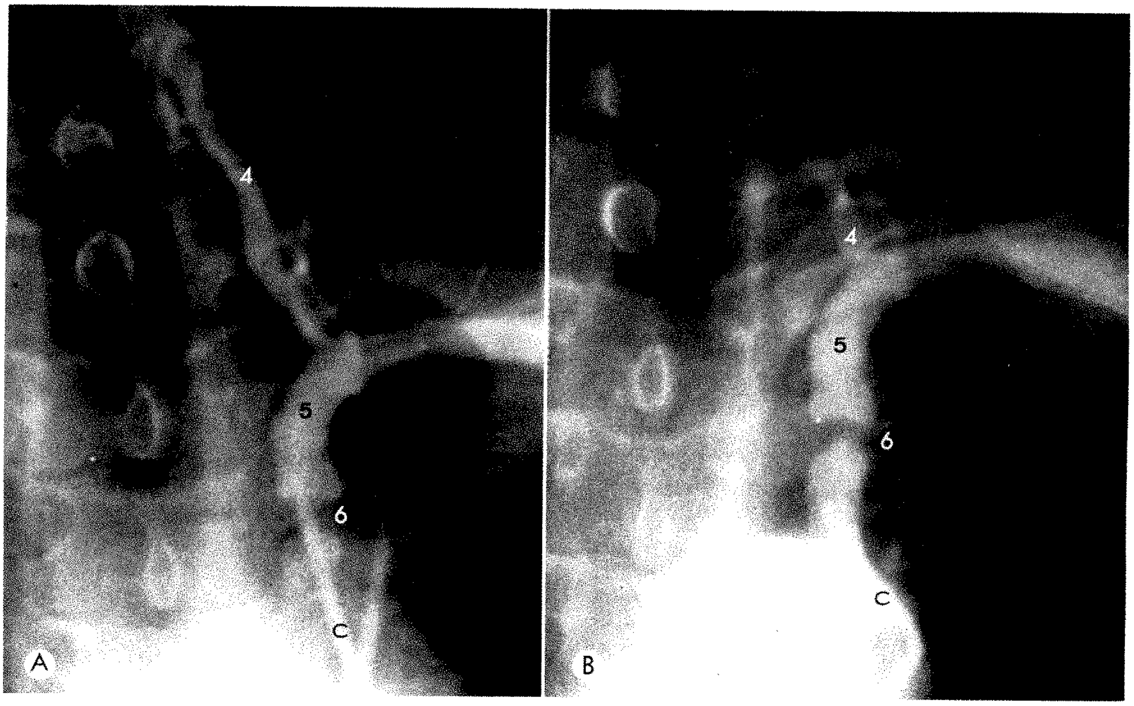

Fig. 6. (*A*) Left subclavian arteriogram. A catheter (C) introduced through the left femoral artery has been advanced until its tip lies distal to the obstruction. The 1 cm. long, sharply localized obstruction (6) in the left subclavian artery (5) lies 3 cm. proximal to the origin of the left vertebral artery (4). The proximal portion of the left vertebral artery has filled by antegrade flow from the subclavian artery, presumably because of injection overpressure.

(*B*) With the catheter tip proximal to the obstruction and at another injection, the small lumen remaining through the obstruction can be seen. Antegrade filling of the left vertebral artery (4) (not well shown in this print) was more pronounced than in the situation illustrated in *A*. Since the additional obstructive influence of the catheter (as in *A*) in producing left subclavian hypotension is now absent, retrograde emptying of the left vertebral artery was even more ineffectual than in the earlier injection (*A*), requiring about 10 seconds for disappearance of contrast material from the vessel. The foregoing observations were established on other test injections and recorded on video tape (not reproduced here).

pressures between the basilar and subclavian arteries to the point where the direction of blood flow is reversed.[12]

SUMMARY

Two unusual examples of the subclavian steal syndrome are presented. The first, probably a patient with Takayasu's syndrome, demonstrated bilateral subclavian steal with additional complete obstruction of the left internal carotid artery and partial obstruction of the right internal carotid artery. The second patient, with incipient subclavian steal due to a partially occluded left subclavian artery (with accompanying complete occlusion of left common carotid artery) shows an unusual etiology for this syndrome, namely, metastatic tumor thrombus.

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CLINICAL USES OF SELECTIVE EXTERNAL CAROTID ARTERIOGRAPHY*

By THOMAS H. NEWTON, M.D., and RICHARD A. KRAMER, M.D.†
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SELECTIVE catheterization of the external carotid artery and its branches improves the accuracy of angiographic diagnosis of certain lesions in the head and neck. As in other peripheral arteriographic examinations, selective opacification of a specific circulatory bed greatly enhances the angiographic detail. In most medical centers the external carotid artery has been studied by arteriography of the common carotid artery. Wickbom[16] and Ruggiero and Jay[11] have described methods of percutaneous puncture of the external carotid artery. The introduction of a needle or cannula from the common carotid artery into the external carotid artery has also been described.[2]

In our experience, percutaneous puncture of the external carotid artery and maintenance of a needle in the lumen have proved difficult. Moreover, by this method the selective opacification of branches of the external carotid artery is impossible. Selective arteriography of the external carotid artery is effectively accomplished by percutaneous catheterization of this artery, as described by Liverud.[9] Weidner and Hanafee[18] and Schechter[12] have pioneered the use of catheter arteriography of the carotid artery and its branches in this country. This method permits a detailed analysis of the external carotid artery and its branches without superimposition of an opacified internal carotid system. Such selective arteriographic study permits better evaluation of both intracranial and extracranial lesions whose blood supply is derived from the external carotid artery. By this method, the nature and extent of the lesion are often accurately analyzed. External carotid arteriography also may aid in planning the surgical approach and in determining the nature of the vascular supply. Moreover, selective percutaneous catheterization of the external carotid artery and its branches may be useful in chemotherapeutic perfusion of neoplasms in the head and neck.[8]

ANATOMY OF THE EXTERNAL CAROTID ARTERY AND ITS BRANCHES

The external carotid artery usually arises from the common carotid artery at the level of the hyoid bone. In its proximal portion it courses anteriorly and medially to the internal carotid artery. The branches of the external carotid artery have been divided into 4 groups: anterior, posterior, ascending, and terminal[4] (Fig. 1).

The anterior branches include the superior thyroid, lingual, and external maxillary arteries. The superior thyroid artery is usually the first branch of the external carotid artery arching downward to supply the thyroid gland and its adjacent muscles. The lingual artery, the second anterior branch of the external carotid artery, initially courses upward and then loops down to supply the tongue. The external maxillary artery arises slightly above the lingual artery and runs anteriorly to the pharynx, tonsil, submaxillary gland, and muscles of the neck and lower face.

Posteriorly, the occipital artery arises at the level of the external maxillary artery, and extends superiorly to supply the muscles in the suboccipital region. Meningeal branches of the occipital artery ascend along the internal jugular vein. They enter the skull through the jugular foramen and condylar canal and supply the dura of the posterior fossa. The posterior auricular artery is the second posterior branch of the external carotid artery. It is a relatively small vessel supplying some of the neck muscles and the parotid gland.

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The ascending pharyngeal artery is a small vessel arising near the origin of the external carotid artery. Posterior meningeal branches arise from this vessel to supply the dura of the posterior fossa. Unless hypertrophied, the meningeal branches of the ascending pharyngeal and occipital arteries are not usually seen on external carotid arteriograms.

The superficial temporal and internal maxillary are the 2 terminal branches of the external carotid artery. The superficial temporal artery appears as a continuation of the external carotid artery. It ends in frontal and parietal branches, which supply the scalp and pericranium. The internal maxillary artery passes forward to supply the deep structures of the face. The middle meningeal artery arises from the internal maxillary artery and enters the skull via the foramen spinosum. It is the largest artery normally supplying the dura mater. After running forward and laterally in a groove in the greater wing of the sphenoid, it divides into anterior and posterior branches. These spread out between the dura and inner table, anastomosing with anterior and posterior meningeal arteries. Normally, the branches of the superficial temporal artery are larger and more tortuous in course than those of the middle meningeal artery. Angiographic identification, therefore, is usually not difficult. When a middle meningeal artery supplying a vascular lesion is hypertrophied, it also becomes more serpiginous. Its identification is aided by recognizing its close relationship to the greater wing of the sphenoid and the pterion and, more distally, to the meningeal vascular grooves of the skull.

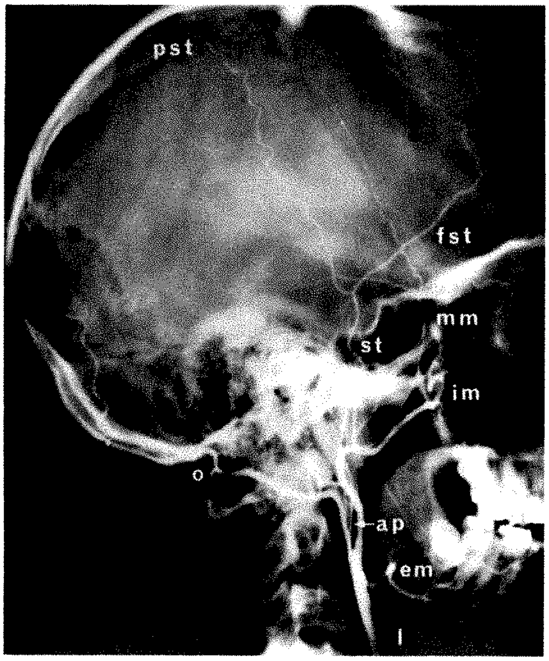


Fig. 1. Arteriogram of normal external carotid artery. l = lingual artery; em = external maxillary artery; o = occipital artery; ap = ascending pharyngeal artery; st = superficial temporal artery; fst = frontal branch of superficial temporal artery; pst = parietal branch of superficial temporal artery; im = internal maxillary artery; mm = middle meningeal artery.

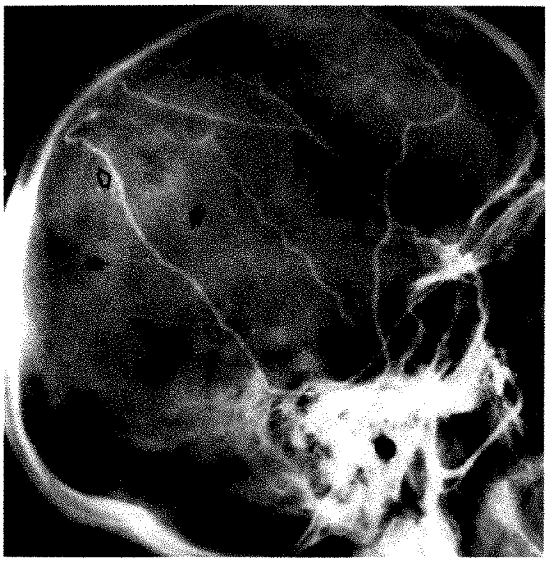
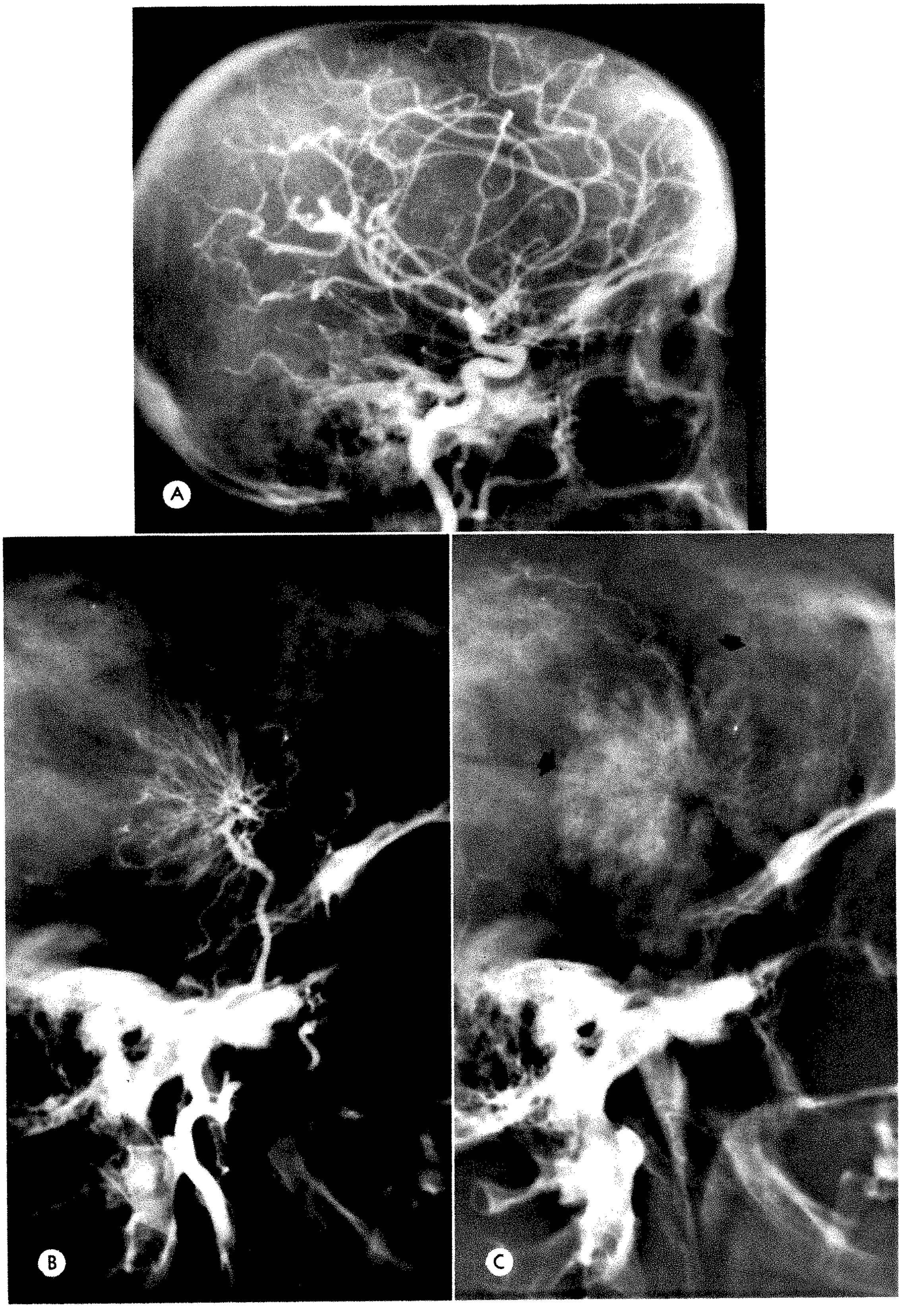


Fig. 2. Large parieto-occipital meningioma. Right external carotid arteriogram shows hypertrophy of the anterior and posterior branches of the middle meningeal artery extending to the region of the tumor. Radiating vessels are seen in the tumor.

TECHNIQUE

The patient is brought to the radiology department in a fasting state. Premedica-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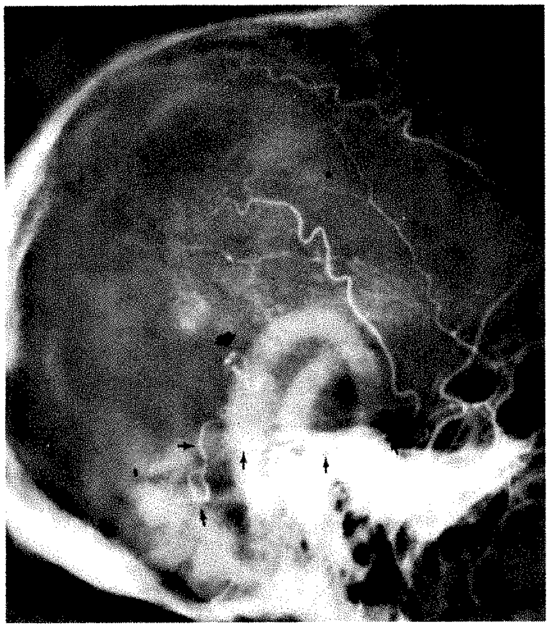

Fig. 4. Occipito-temporal glioma with invasion of tentorium. Selective external carotid arteriogram shows the posterior branch of the middle meningeal artery ending in a tangle of abnormal vessels (arrows). Selective internal carotid arteriogram had shown an enlarged tentorial branch extending posteriorly and ending in a tumor blush. The roentgenologic diagnosis of tentorial meningioma was disproved at surgery where a glioma infiltrating the tentorium was found.

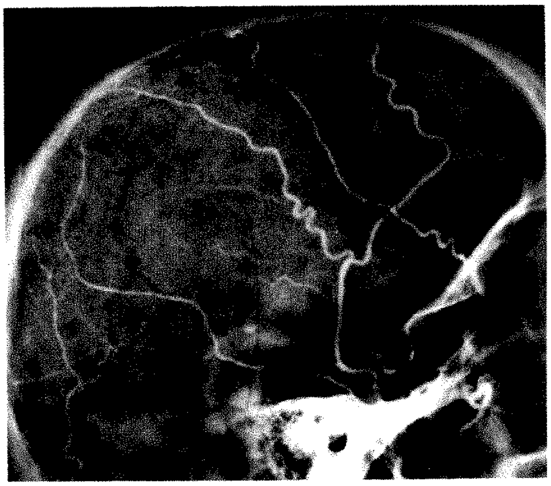

Fig. 5. Subdural lymphoma. This was a 40 year old white woman with recent onset of sensory and motor seizures involving the left lower extremity. No abnormalities were noted on physical examination. Selective external carotid arteriography showed hypertrophied branches of the middle meningeal artery supplying an extracerebral tumor in the right parietal region, high on the convexity. At craniotomy, a well circumscribed, subdural lymphosarcoma invading the adjacent dura was excised.

tion in the adult consists of benadryl 50 mg., and nembutal, 50 mg. intramuscularly, 30 minutes before the procedure. Atropine 0.4 mg. is given intramuscularly just before the study is begun. After infiltration with a local anesthetic, a small incision is made in the skin, at a point low in the neck, to facilitate later passage of the catheter. The common carotid artery is then punctured with an 18 gauge thin-walled needle. The carotid bifurcation is visualized in the lateral projection, using Polaroid film and a test injection of 3 to 4 cc. of contrast material. This preliminary roentgenogram

Fig. 3. Large frontotemporal meningioma arising in the region of the pterion. (*A*) Left common carotid arteriogram shows distortion of the sylvian triangle, but no definite pathologic vessels are identified. (*B*) Selective external carotid arteriogram (arterial phase). Enlarged middle meningeal artery ending in a cluster of tumor vessels with a radial spoke-like pattern. (*C*) External carotid arteriogram (capillary phase). A large vascular blush indicates the size of the tumor.

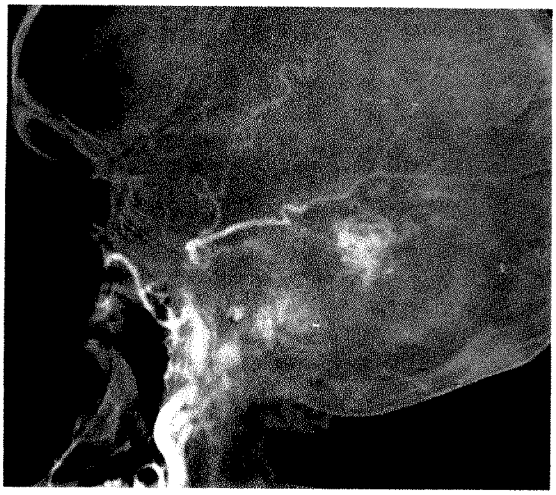

Fig. 6. Metastatic neoplasm of the skull from carcinoma of the thyroid. This 76 year old woman had had a thyroidectomy for carcinoma in 1951. Since 1958 a progressively enlarging osteolytic lesion had been noted in the right parietal bone with an overlying soft tissue mass. Selective external carotid arteriogram (subtraction technique) made in 1965 showed an enlarged parietal branch of the superficial temporal artery ending in a large vascular tumor blush in the region of the palpable soft tissue mass and osteolytic skull lesion.

verifies proper placement of the needle and also demonstrates its relationship to the carotid bifurcation.

Catheterization is not performed when significant atherosclerotic changes are present at the bifurcation, since such manipulation could dislodge atheromatous material. In these instances, arteriography is performed by injection of contrast material through the needle into the common carotid artery. When the bifurcation appears normal, a wire guide is carefully advanced through the needle into the vessel for a distance of 5 cm. The guide measures .035

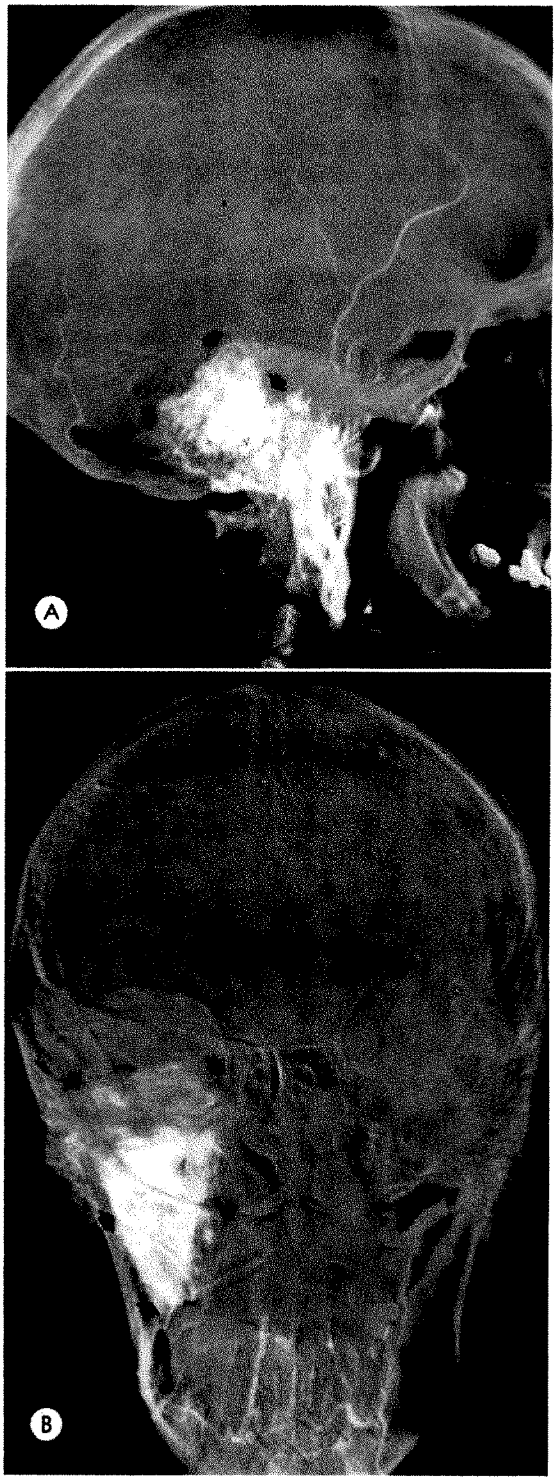

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Fig. 7. (*A* and *B*) Glomus jugulare tumor. A 34 year old woman with progressive tinnitus and deafness in the right ear. Selective external carotid arteriography (subtraction technique) showed extensive vascular blush in the region of the petrous bone. Abnormal tumor vessels were also seen on selective vertebral arteriography.

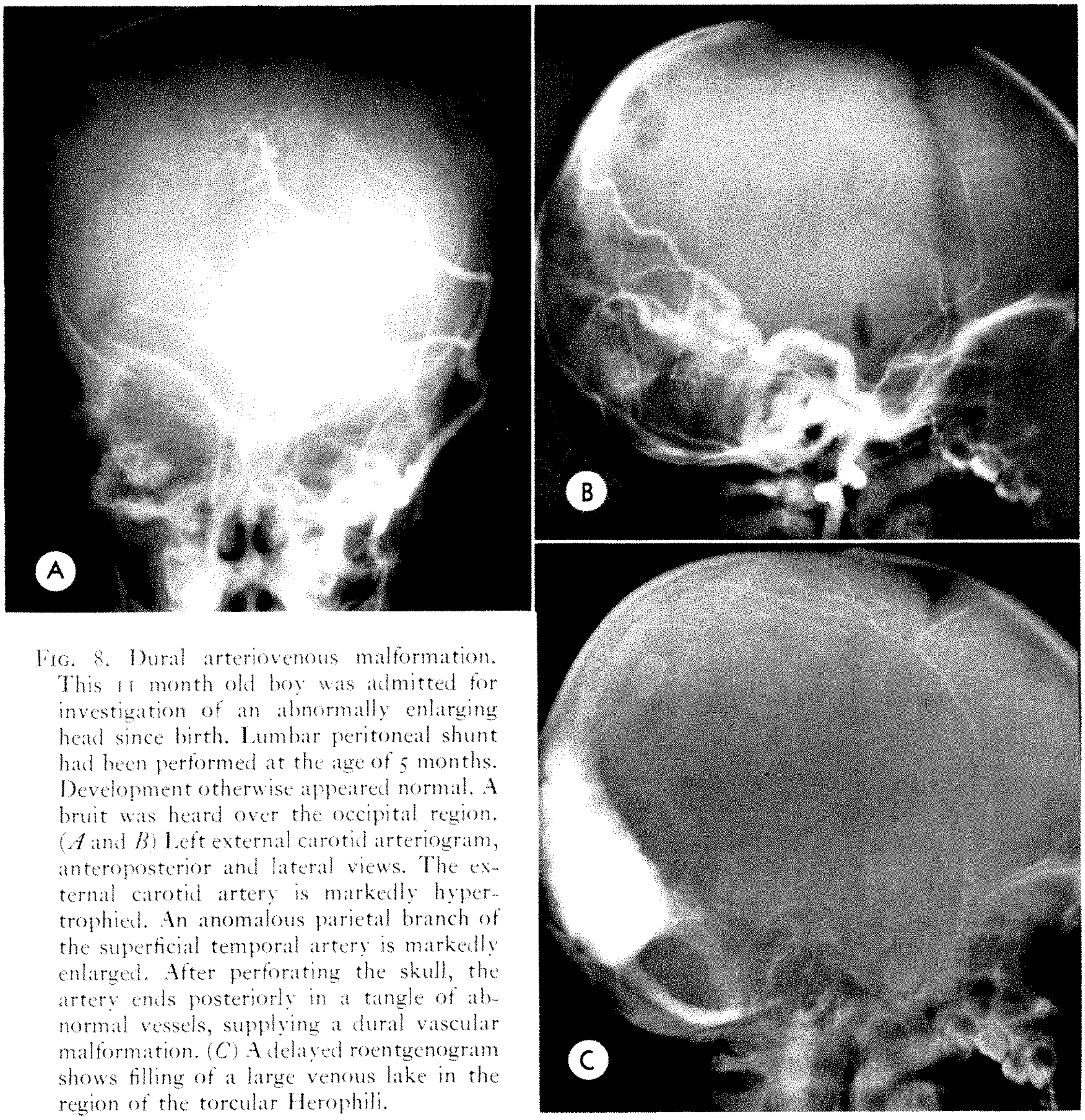

FIG. 8. Dural arteriovenous malformation. This 11 month old boy was admitted for investigation of an abnormally enlarging head since birth. Lumbar peritoneal shunt had been performed at the age of 5 months. Development otherwise appeared normal. A bruit was heard over the occipital region. (*A* and *B*) Left external carotid arteriogram, anteroposterior and lateral views. The external carotid artery is markedly hypertrophied. An anomalous parietal branch of the superficial temporal artery is markedly enlarged. After perforating the skull, the artery ends posteriorly in a tangle of abnormal vessels, supplying a dural vascular malformation. (*C*) A delayed roentgenogram shows filling of a large venous lake in the region of the torcular Herophili.

inch in diameter and has a 2 cm. flexible tip. The needle is then withdrawn and a preshaped, tapered, radiopaque polyethylene catheter is advanced over the wire and threaded into the artery. The catheter is 40 cm. in length, .065 inch in outer diameter and .045 inch in inner diameter.* Its tip is slightly curved and the catheter is marked with a laundry marking pen at a point 10 cm. from the tip. Care is exercised to avoid passage of the wire guide or the catheter beyond the 10 cm. level. In children the technique is similar except that a 19 gauge thin-walled needle is used. The wire guide measures .025 inch in diameter and the catheter† measures .037 inch in inner diameter.

The position of the catheter is checked by using a small amount of contrast medium and a horizontal image amplifier. If the catheter tip has entered the internal carotid artery, it is gently withdrawn under horizontal fluoroscopic guidance. During its slow retraction, the curved tip of the

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* Becton and Dickinson RPX 045-H.

† Becton and Dickinson RPX 037-H.

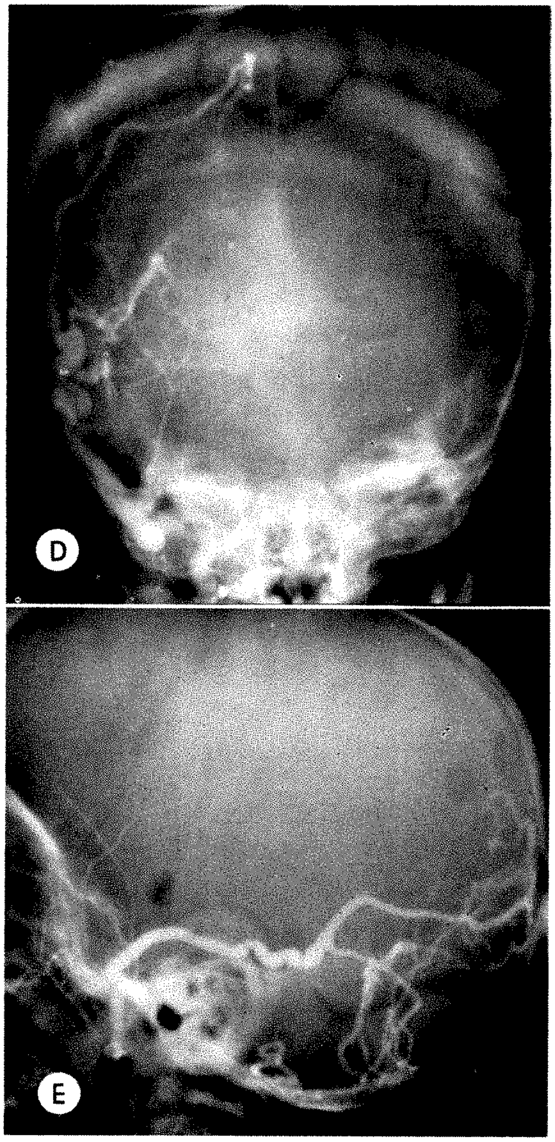

FIG. 8. (*D* and *E*) Selective right external carotid arteriogram. Anteroposterior and lateral roentgenograms show similar findings on the right side.

catheter is pointed anteriorly and will be seen to flip into the external carotid artery. The catheter is then advanced into the external carotid artery and is usually placed just distal to the origin of the lingual artery. If desirable, however, the tip of the catheter can be placed selectively into the major branches of the external carotid artery. By extending the patient's neck, the external carotid artery is in line with the common carotid artery; this extension facilitates placement of the wire guide and catheter into the external carotid artery.

When contrast medium is injected into the external carotid artery, the patient usually experiences a feeling of heat in the face on the side of injection. With internal carotid placement, this sensation is usually noted behind the eye. Moreover, with an injection of saline into the external carotid artery, unilateral blanching of the face and cheek is often observed. With injection into the internal carotid artery, blanching occurs along the medial aspect of the orbit and the adjacent forehead. To prevent clotting and dislodgment of emboli, the catheter is aspirated frequently and flushed with heparinized saline at least once each minute.

We have also used the transfemoral approach to selective catheterization of the external carotid artery. This method has been particularly useful when arteriog-

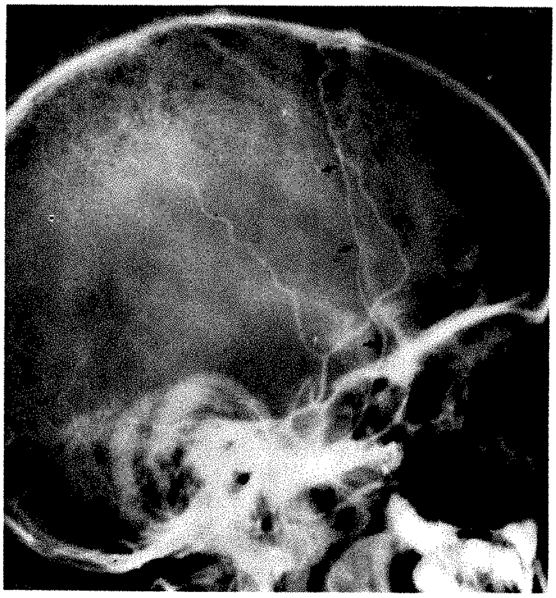

FIG. 9. Extensive intracerebral arteriovenous malformation. Selective external carotid arteriogram shows prominent anterior branch of middle meningeal artery extending anteriorly. This vessel ends in a small tangle of abnormal vessels in the frontal region (large arrow). Selective internal carotid arteriogram (not shown here) revealed extensive arteriovenous malformation involving the frontal lobe and basal ganglia.

raphy of both carotid and vertebral systems has been indicated.

Usually, serial roentgenograms are obtained in lateral projections although in selected cases anteroposterior, submentovertical, or other projections are helpful. Our patients have tolerated a 60 per cent solution of conray* particularly well, and in the adult 5 cc. is introduced within 1.5 to 3 seconds by hand injection. Too rapid injection will result in reflux of contrast medium into the internal carotid artery.

The complications of carotid catheterization are related primarily to vessel damage during introduction and manipulation of the needle before catheter insertion. These complications are similar to hotse of simple percutaneous puncture of the carotid artery. Catheterization adds the potential hazard of arterial spasm should the catheter or wire guide be advanced too far or too forcefully.[12] Although the advancing catheter or guide wire can dislodge thrombi or atheromatous plaques, the preliminary roentgenogram showing the bifurcation markedly reduces this risk. Intravascular breakage of wire guides has been described in other Seldinger catheter replacement techniques and is a potential complication at this site as well.[3] All these complications are avoidable by the experienced worker using careful technique.

A great advantage of the catheter technique is the avoidance of subintimal or extravascular injection. It also facilitates positioning of the patient. The relative safety of this approach in experienced hands is reflected by the few complications in the large series of carotid catheterizations reported by Amundsen *et al.*,[1] Liverud,[9] and Schechter.[12]

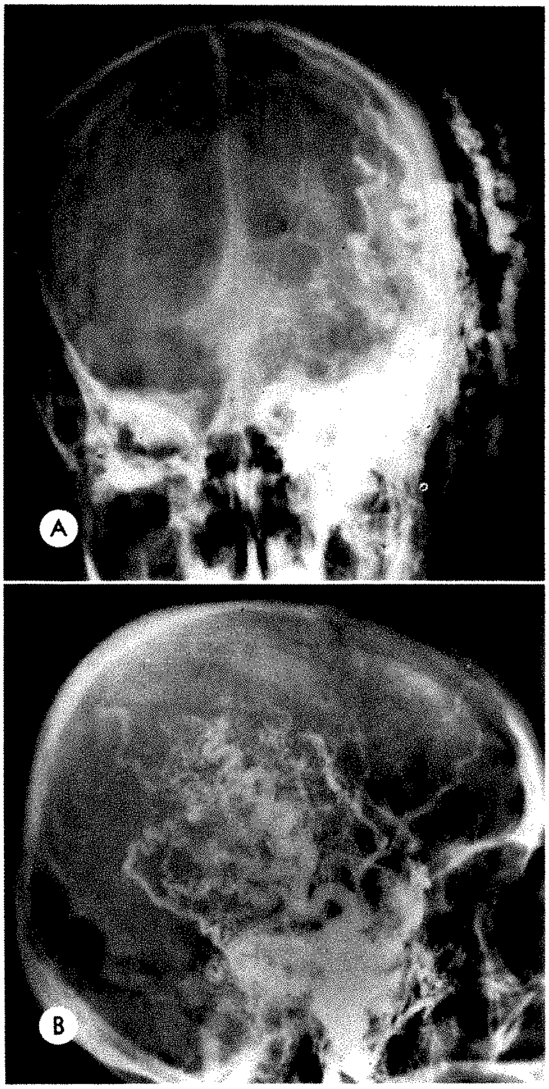

Fig. 10. (*A* and *B*) Cirsoid aneurysm of the scalp. This 35 year old woman had noted a raised reddish area in front of the left ear. Biopsy showed a hemangioendothelioma. After the biopsy, the mass in the left scalp grew rapidly. Selective left external carotid arteriogram showed marked hypertrophy of the superficial temporal branches with large tortuous irregular vessels in the scalp, defining the extent and vascularity of the lesion.

CLINICAL MATERIAL

INTRACRANIAL LESIONS

Meningiomas, the most common intracranial and extracerebral neoplasms, usually have a dual circulation with contributions from both the internal and external carotid arteries.[17] Usually, the middle meningeal artery supplies these tumors (Fig. 2), although, depending on their location, other smaller meningeal branches may also contribute to the blood supply.[13] The branches of the external carotid artery supplying the tumor are enlarged and usually abnormally tortuous. The vessels within the tumor itself are tortuous but their lu-

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* Mallinckrodt Chemical Works, St. Louis, Missouri.

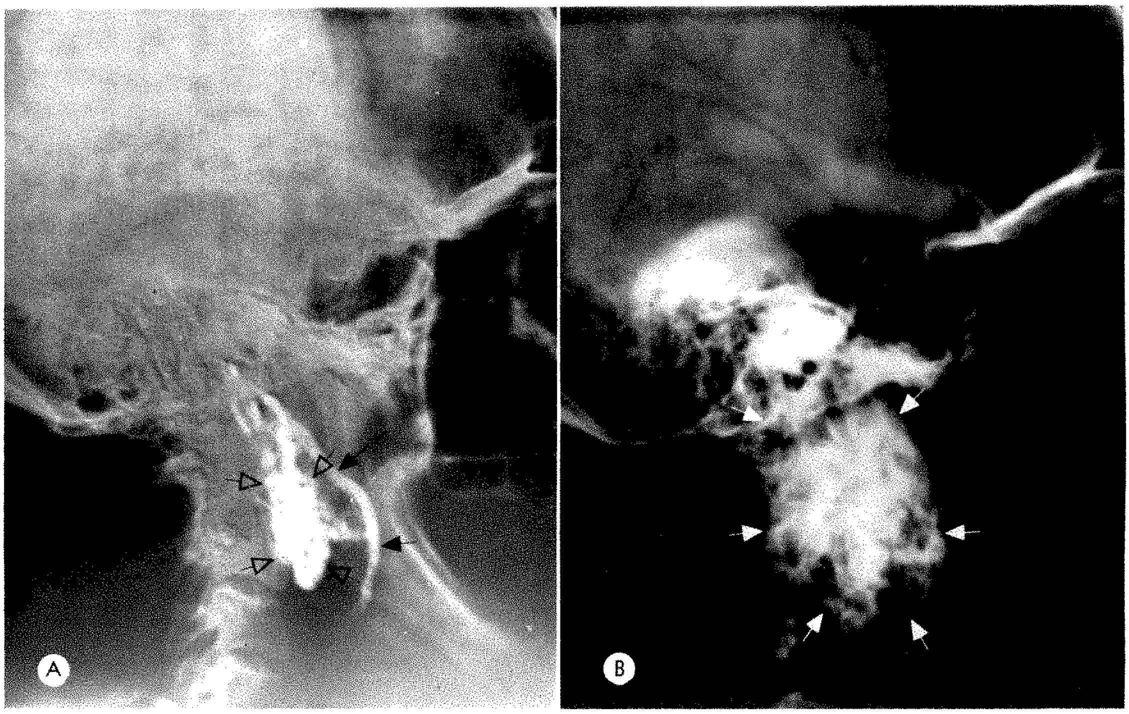

FIG. 11. Glomus vagale tumor. An asymptomatic mass displacing the tonsillar region medially was discovered in a 54 year old woman on routine physical examination. External carotid arteriography showed a large vascular mass in the right tonsillar region. At operation a 5×3 cm. tumor was found to extend from the jugular fossa into the neck. The vagus coursed directly through the tumor. (*A*) External carotid arteriogram, arterial phase. The external carotid artery (solid arrows) is bowed forward by a vascular tumor. (*B*) Vascular blush in the venous phase defines the full extent of the tumor.

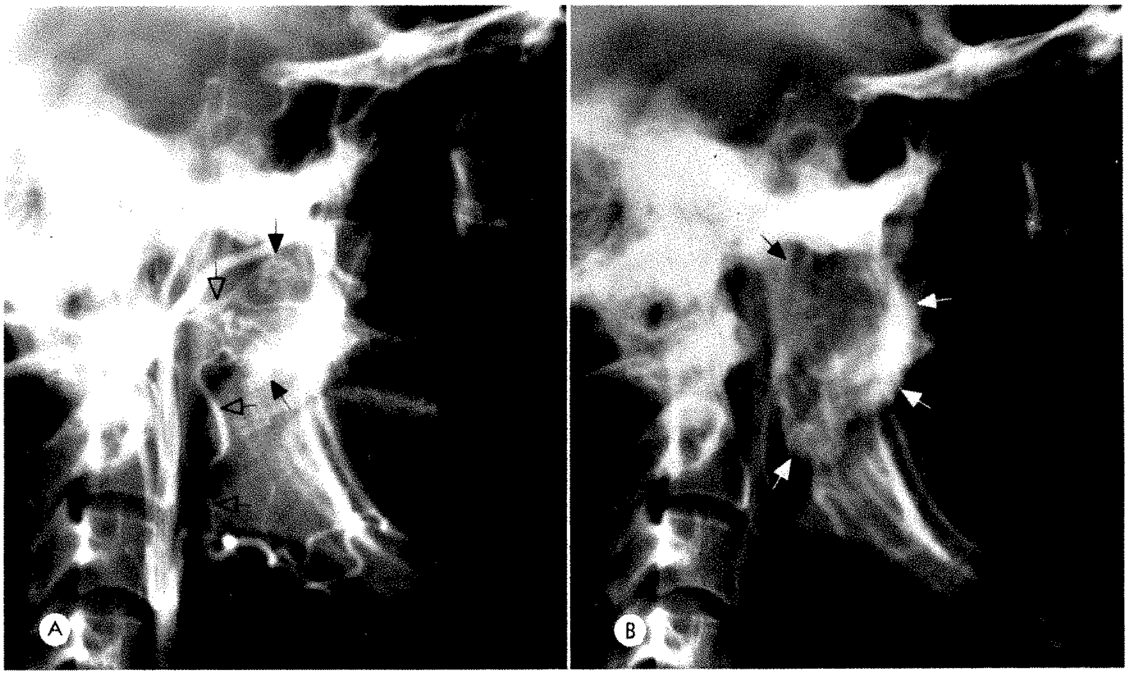

FIG. 12. Nasopharyngeal fibroma. A 28 year old man with a 6 months' history of nasal obstruction and rhinorrhea. (*A*) External carotid arteriogram, arterial phase. Hypertrophied branches of the internal and external maxillary arteries (open arrows) end in a tangle of abnormal vessels (solid arrows) in the posterior nasopharynx. (*B*) Venous phase showing the tumor outlined by a vascular blush.

mina regular. The tumor vessels frequently have a radial spoke-like appearance. Numerous capillaries are often present, which remain filled late into the venous phase, giving a prolonged homogeneous stain. Only the central portions of the tumor may be demonstrated by external carotid arteriography, whereas the peripheral portions may be opacified by internal carotid arteriography.[5] When adjacent bone or soft tissue is involved, the superficial temporal artery, occipital artery, or other branches of the external carotid artery may contribute to the tumor circulation as well.

When common carotid arteriography is performed, the contribution of the external carotid artery may not be appreciated. An example of such a situation is illustrated in Figure 3, *A*, *B* and *C*. In this instance, the extracerebral nature of the tumor was not recognized until selective external carotid arteriography was performed.

Other intracranial neoplasms which involve the meninges may derive part or all of their blood supply from the external carotid system. Invasion of dura by gliomas is rare. In these rare instances, however, external carotid arteriography may demonstrate vascular contribution to the tumor from this system (Fig. 4).

We have studied one patient with a subdural lymphoma in whom the external carotid arteriogram indicated the extracerebral nature of the process (Fig. 5). Metastatic neoplasms to the skull may occasionally be demonstrated by external carotid arteriography (Fig. 6).

Glomus jugulare tumors are usually found in the jugular fossa, causing enlargement of the jugular fossa and destruction of the adjacent temporal bone. A significant portion of their blood supply is derived from the external carotid system. Thus, external carotid arteriography is helpful in determining the nature of the tumor as well as in assessing its extent and the origin of its blood supply (Fig. 7, *A* and *B*).

Vascular malformations involving the dura are supplied, at least in part, by the external carotid artery and are well demonstrated by selective arteriography (Fig. 8, *A–E*). In these cases, selective opacification of the internal carotid, the external carotid, and vertebral arteries defines the vascular pattern more precisely than does nonselective arteriography. Surgical treatment is thus facilitated.

Occasionally, intracerebral arteriovenous malformations may be opacified by external carotid arteriography since they may derive part of their blood supply from the external as well as the internal carotid arteries (Fig. 9). The external carotid arterial contribution may be a reflection of associated dural vascular malformations, or may represent contributory circulation via the leptomeningeal and rete mirabile anastomoses.[19]

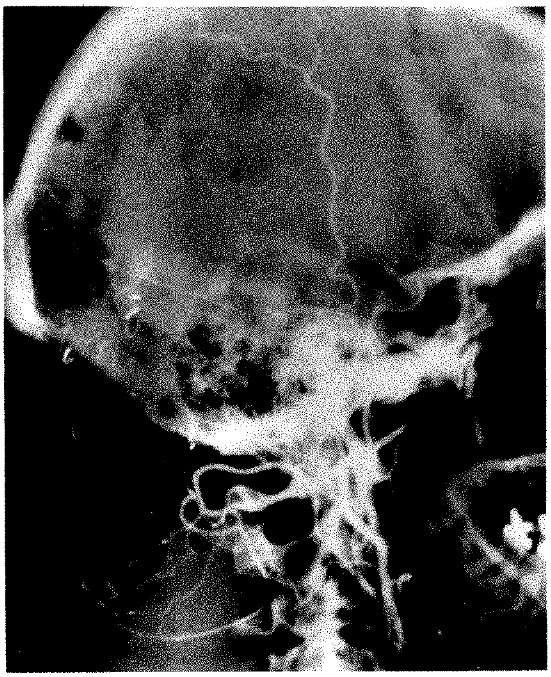

FIG. 13. Recurrent tentorial meningioma extending into the neck. This 55 year old woman had had a suboccipital craniotomy in 1956 for a tentorial meningioma. She was readmitted 8 years later because of a large soft tissue mass in the suboccipital region. Selective external carotid arteriogram showed a large, vascular, suboccipital soft-tissue mass largely supplied by the occipital branch of the external carotid artery. Selective vertebral arteriography also showed contribution from muscular branches of the vertebral artery.

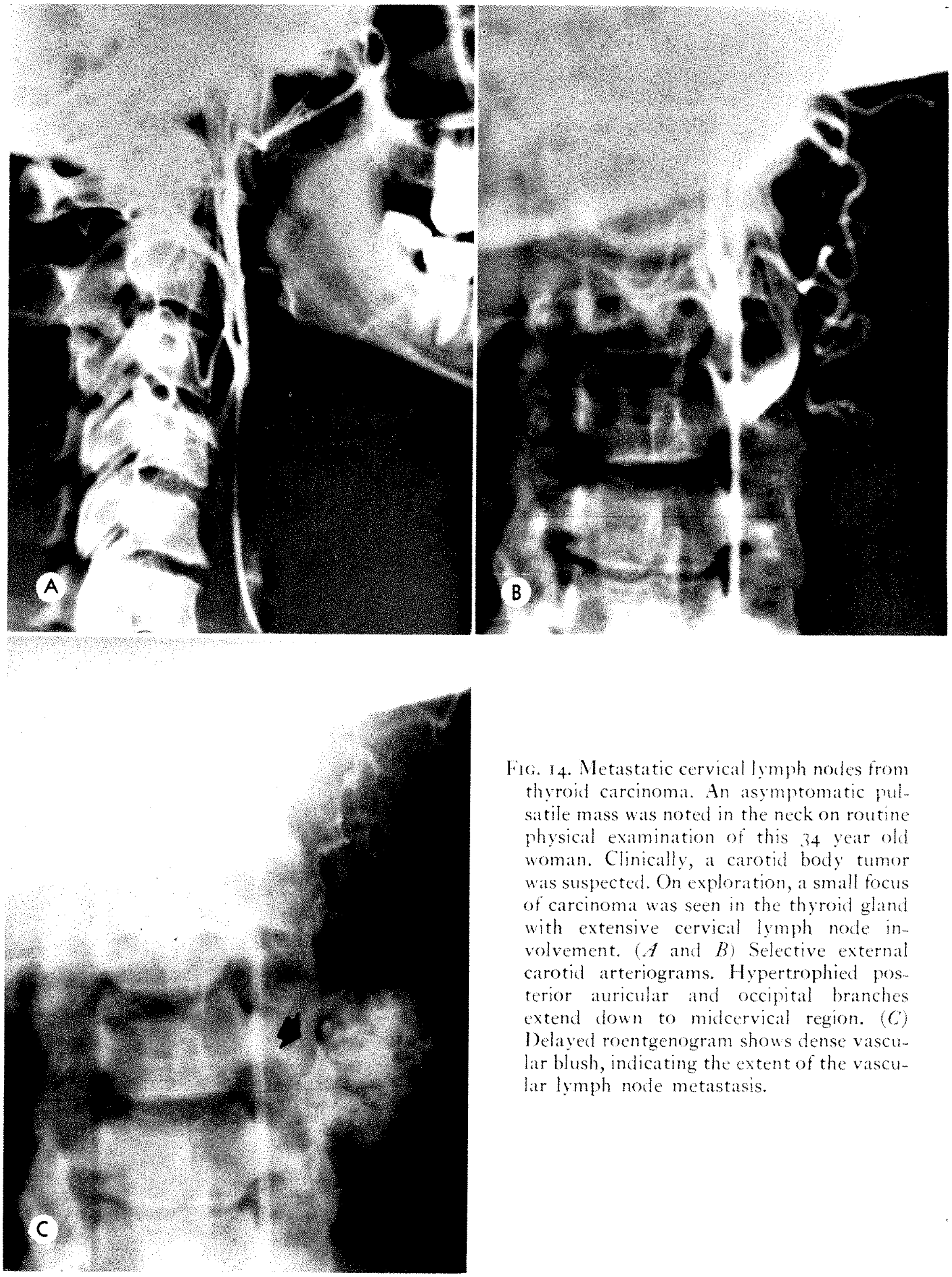

Fig. 14. Metastatic cervical lymph nodes from thyroid carcinoma. An asymptomatic pulsatile mass was noted in the neck on routine physical examination of this 34 year old woman. Clinically, a carotid body tumor was suspected. On exploration, a small focus of carcinoma was seen in the thyroid gland with extensive cervical lymph node involvement. (*A* and *B*) Selective external carotid arteriograms. Hypertrophied posterior auricular and occipital branches extend down to midcervical region. (*C*) Delayed roentgenogram shows dense vascular blush, indicating the extent of the vascular lymph node metastasis.

EXTRACRANIAL LESIONS

The extent of cirsoid aneurysms, hemangiomas, and vascular malformations involving the scalp is best evaluated by external carotid arteriography. In these cases marked hypertrophy of the feeding branches of the external carotid artery may be observed (Fig. 10, *A* and *B*).

Angiographic investigation of neoplasms of the neck may establish the nature and extent of the lesion. Chemodectomas, such as carotid body and glomus jugulare tumors, may occur along the course of the carotid artery in the neck (Fig. 11, *A* and *B*). Carotid body tumors are vascular lesions which can only be properly evaluated by selective arteriography. These tumors usually are located at the bifurcation of the common carotid artery and thus cause a separation of the proximal internal and external carotid arteries. Since carotid body tumors usually receive their major blood supply from the external carotid artery, selective external carotid arteriography will demonstrate a plethora of tumor vessels just distal to the bifurcation of the common carotid artery. Since the thyrocervical trunk, costocervical trunk, and vertebral artery may contribute to these tumors, however, angiography of these vessels is recommended for more complete preoperative evaluation.[19] Complete preoperative arteriographic studies are particularly important in view of the high morbidity and mortality associated with surgical therapy of these extremely vascular tumors.[15]

Juvenile nasopharyngeal fibromas are uncommon, very vascular tumors (Fig. 12, *A* and *B*), usually seen in adolescent boys and, occasionally, in young women. Holman and Miller[7] have recently emphasized characteristic plain roentgenographic findings, which in conjunction with the clinical picture, can lead to a correct preoperative diagnosis. Because of their extreme vascularity, surgical treatment is fraught with difficulty, and the surgeon can be aided considerably by arteriographic studies. Although the major vascular supply is from the external carotid artery, contributions may be from the internal carotid as well.[14]

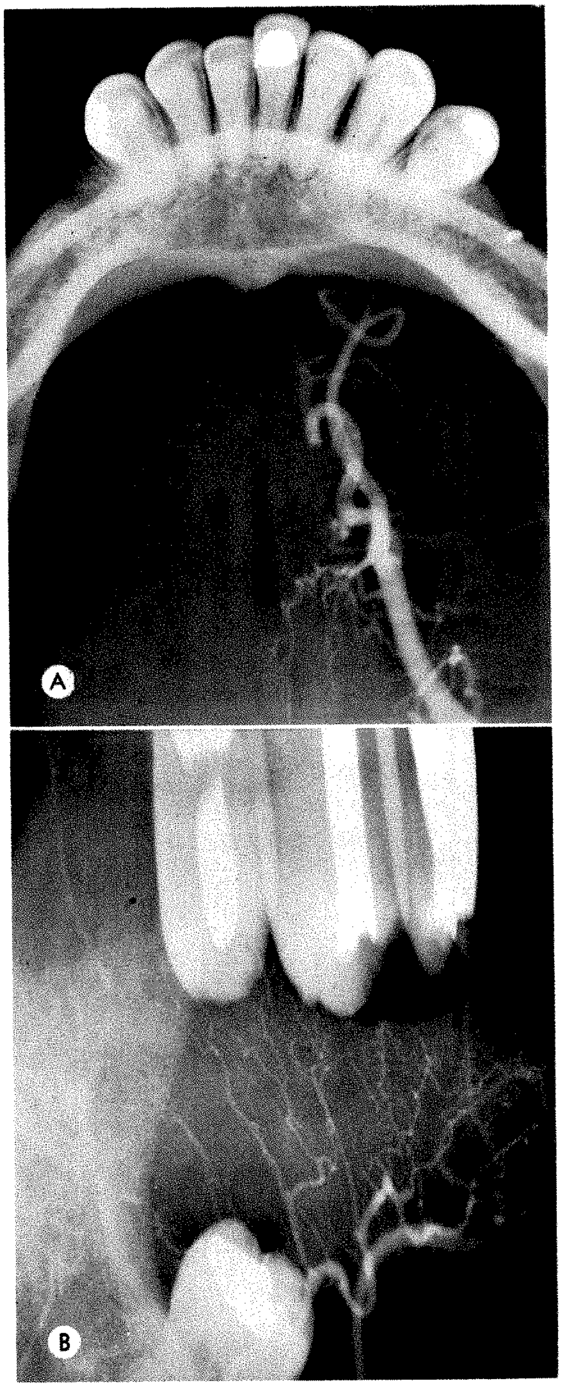

Fig. 15. Lingual arteriography. (*A* and *B*) Anteroposterior and lateral roentgenograms of the tongue show a normal vascular pattern.

The external carotid artery may supply

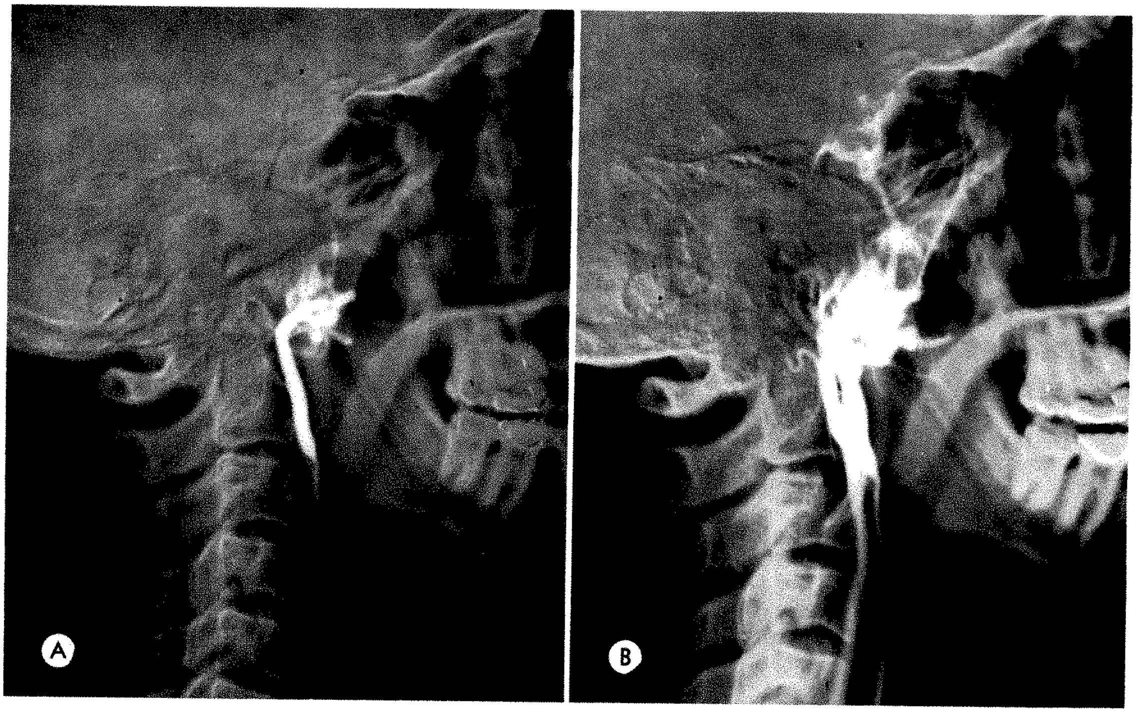

FIG. 16. (*A* and *B*) Traumatic arteriovenous fistula of the external carotid artery. This was an 18 year old man who was stabbed in the cheek. Shortly after the injury, a rhythmic hissing sound was noted by the patient. On physical examination a bruit in the neck was noted, which was stopped by carotid compression. Selective external carotid arteriography (subtraction technique) showed immediate filling of the pterygoid venous plexus and early venous drainage (arrows).

intracranial neoplasms which extend into the neck and external carotid arteriography in these cases is a useful diagnostic procedure. An example of a meningioma which extended from the posterior fossa into the neck is illustrated (Fig. 13). Similarly, the extent and vascularity of metastatic neoplastic masses in the neck may be evaluated by external carotid angiography (Fig. 14, *A*, *B* and *C*).

The arteriographic pattern of neoplasms that involve the tongue may be studied in detail by means of selective external carotid or lingual arteriography (Fig. 15, *A* and *B*). Angiography of the tongue, using non-screen, bone-free films, would allow a detailed analysis of tumor vessels, both before and after therapy.

Traumatic arteriovenous fistulas of the external carotid artery are not common but are seen following missile or stab wounds. In these cases selective external carotid arteriography establishes the exact location as well as the venous drainage of such a lesion (Fig. 16, *A* and *B*).

As yet, we have not had the opportunity to perform selective external carotid arteriography in patients with epidural hematomas. In these cases, leakage of contrast medium from the ruptured meningeal arteries and displacement of the artery away from the inner table have been described.[10]

A pitfall to be avoided in selective arteriography is the placement of the catheter beyond the origin of the branch supplying the lesion, which may result in failure to opacify the abnormal circulation (Fig. 17, *A*, *B* and *C*).

CONCLUSION

Selective external carotid arteriography, using the catheter technique, represents a relatively simple method for detailed analysis of the external carotid artery and its branches. This method of investigation is

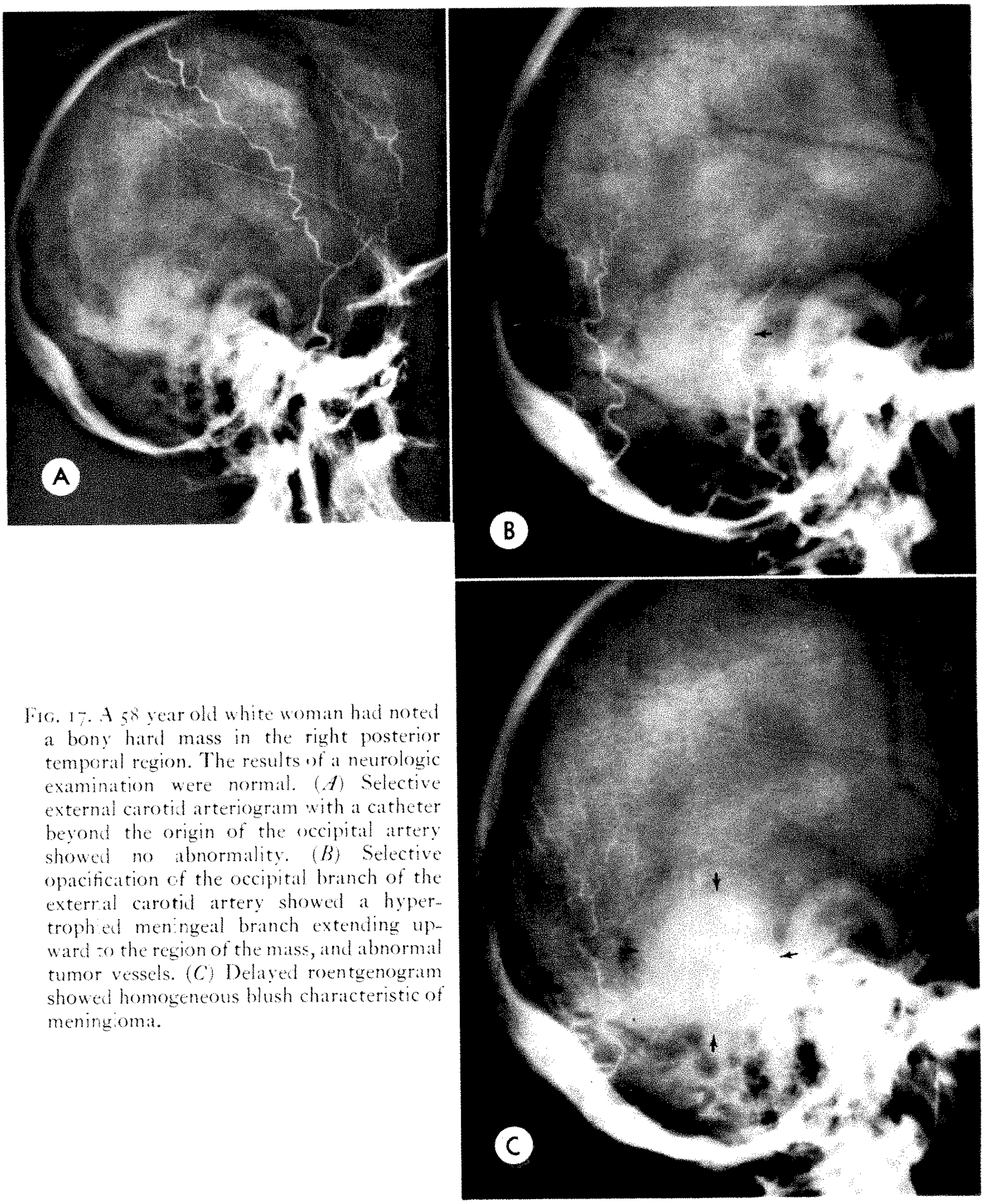

Fig. 17. A 58 year old white woman had noted a bony hard mass in the right posterior temporal region. The results of a neurologic examination were normal. (*A*) Selective external carotid arteriogram with a catheter beyond the origin of the occipital artery showed no abnormality. (*B*) Selective opacification of the occipital branch of the external carotid artery showed a hypertrophied meningeal branch extending upward to the region of the mass, and abnormal tumor vessels. (*C*) Delayed roentgenogram showed homogeneous blush characteristic of meningioma.

valuable in assessing the nature and extent of a variety of vascular lesions of the head and neck. Illustrative examples are presented.

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DEVELOPMENT OF COLLATERAL VESSELS IN AN ACUTE OCCLUSION OF THE COMMON HEPATIC ARTERY*

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THAT the liver can withstand occlusion of the common hepatic artery proximal to the origin of the gastroduodenal artery has been demonstrated in animals[2,3] and in man.[1] Michels[4] has listed 26 potential collateral pathways which may develop to supply a liver so deprived of its arterial blood supply. Only a few of these pathways, however, actually develop to a degree which is angiographically demonstrable. In a recent review of the angiographic findings in long-standing celiac stenosis,[5] it was observed that, in the absence of liver diseases which cause increased demand for arterial blood flow or vascular anomalies, the only collaterals which developed to the liver were the arteries in the head of the pancreas and the gastroduodenal artery. An unusual complication of selective celiac angiography, subintimal injection, has provided the opportunity to observe the immediate collateral development following occlusion of the common hepatic artery.

REPORT OF A CASE

A 65 year old male was admitted to the hospital because of repeated attacks of severe upper abdominal pain. Pancreatic function tests showed decreased enzyme production, and he was considered to have pancreatitis. To exclude a pancreatic neoplasm, a combined celiac-superior mesenteric angiographic study was made.

Angiography was performed through two end-open, yellow KIFA catheters, one introduced from each femoral artery. As the position of the catheters was monitored with small injections of contrast medium during the catheterization procedure, it was shown that the splenic and hepatic arteries had separate origins from the aorta. For the combined injection, therefore, one catheter was introduced into the origin of the common hepatic artery, and the other, into the superior mesenteric artery. An injection of 45 cc. of 76 per cent urografin (Schering, Germany) was made through both catheters simultaneously using a Y-connector with an injection rate of 18 cc. per second. A series of exposures was made in the anteroposterior position using an Elema roll film changer. The patient was then rotated into a right posterior oblique position, and a second injection was made. The catheter was removed from the common hepatic orifice and placed in the splenic orifice, and a single injection of 30 cc. of 76 per cent urografin was made through this catheter in the anteroposterior projection. The catheters were then withdrawn and the patient was returned to the ward. The patient experienced no unusual sensations during or after the procedure. He was pain free at the time of the examination and remained so for several weeks. When his pain returned, it was the same as the pain for which he had originally been admitted.

ANGIOGRAPHIC FINDINGS

In the anteroposterior series of the combined study (Fig. 1), there was good filling of both the hepatic and superior mesenteric arteries and their branches. No evidence of pancreatitis or tumor was noted. In the oblique series of the combined study (Fig. 2, *A*, *B* and *C*), the common hepatic artery was irregularly occluded just proximal to the origin of the gastroduodenal artery by a subintimal injection. The superior mesenteric artery and its branches were normally filled. The inferior pancreaticoduodenal artery, arising from the first jejunal artery, and the anterior pancreaticoduodenal arcade were densely opacified and had increased in diameter approximately 3 times compared with the series taken prior to

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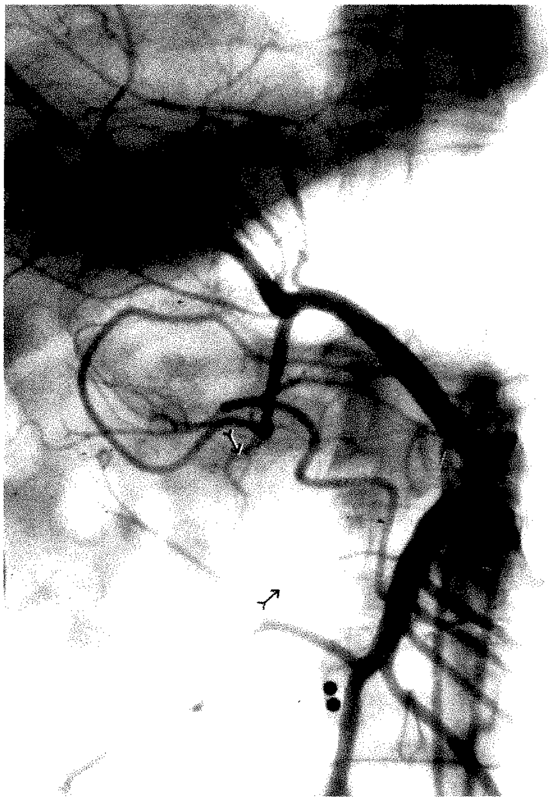

FIG. 1. Combined injection of the common hepatic and superior mesenteric arteries in the anteroposterior position. No abnormalities are seen. The anterior pancreaticoduodenal arcade (↣) is normal in size, and the branches of the dorsal pancreatic artery in the head of the pancreas are not seen.

occlusion. A flow defect caused by nonopacified blood from the posterior pancreaticoduodenal arcade was seen in the mid-portion of the gastroduodenal artery. The proper hepatic artery was filled from the gastroduodenal artery. The right gastric artery could not be seen in this study. At the splenic artery injection (Fig. 3), branches of the dorsal pancreatic artery, which arose from the splenic artery, were larger than they are usually seen to be. The retroduodenal branch and its branch to the uncinate process were particularly prominent. These and other smaller branches of the dorsal pancreatic artery in the head of the pancreas supplied collateral flow to the proper hepatic artery via the posterior pancreaticoduodenal arcades and gastroduodenal artery. The branches of the dorsal pancreatic artery, unfortunately, were not filled in the series taken prior to occlusion, and no comparison could be made. Unopacified blood from the anterior arcade mixed in the gastroduodenal artery. The splenic and left gastric arteries and their branches were normal in appearance.

DISCUSSION

Although Michels[4] listed 26 potential collateral pathways to the liver, 10 of these are anatomic variations of the hepatic artery. In a given person, therefore, 16 or 17 pathways actually exist. In only 7 of these are the communications between hepatic and nonhepatic vessels large enough to provide an immediate collateral supply after a hepatic occlusion. The other 9 are very small communications, either between the vasa vasorum of hepatic and nonhepatic arteries, between duodenal and hepatic arterial branches in the hepatic duct walls, or between branches of extra-abdominal arteries and hepatic artery branches in the supporting ligaments of the liver. These are too small to develop immediately as collaterals, and probably develop only in long standing occlusions. Of the 7 large collateral pathways, the gastroduodenal artery is the final vessel in 5, the other 2 sizable collateral systems occurring between branches of the left gastric and hepatic arteries.

In this patient, the acute collateral blood supply to the liver occurred entirely over the arteries in the head of the pancreas. Three other collateral pathways which might have been expected to develop immediately were not seen. The first is from the splenic artery via the left and right gastro-epiploic arteries to the gastroduodenal artery, and the second, from the splenic artery via the inferior epiploic arcade to the gastroduodenal artery. The third is from the left gastric artery via the right gastric artery to the right hepatic artery. That these 3 pathways did not develop in this patient is probably related to the very small size of the right gastric

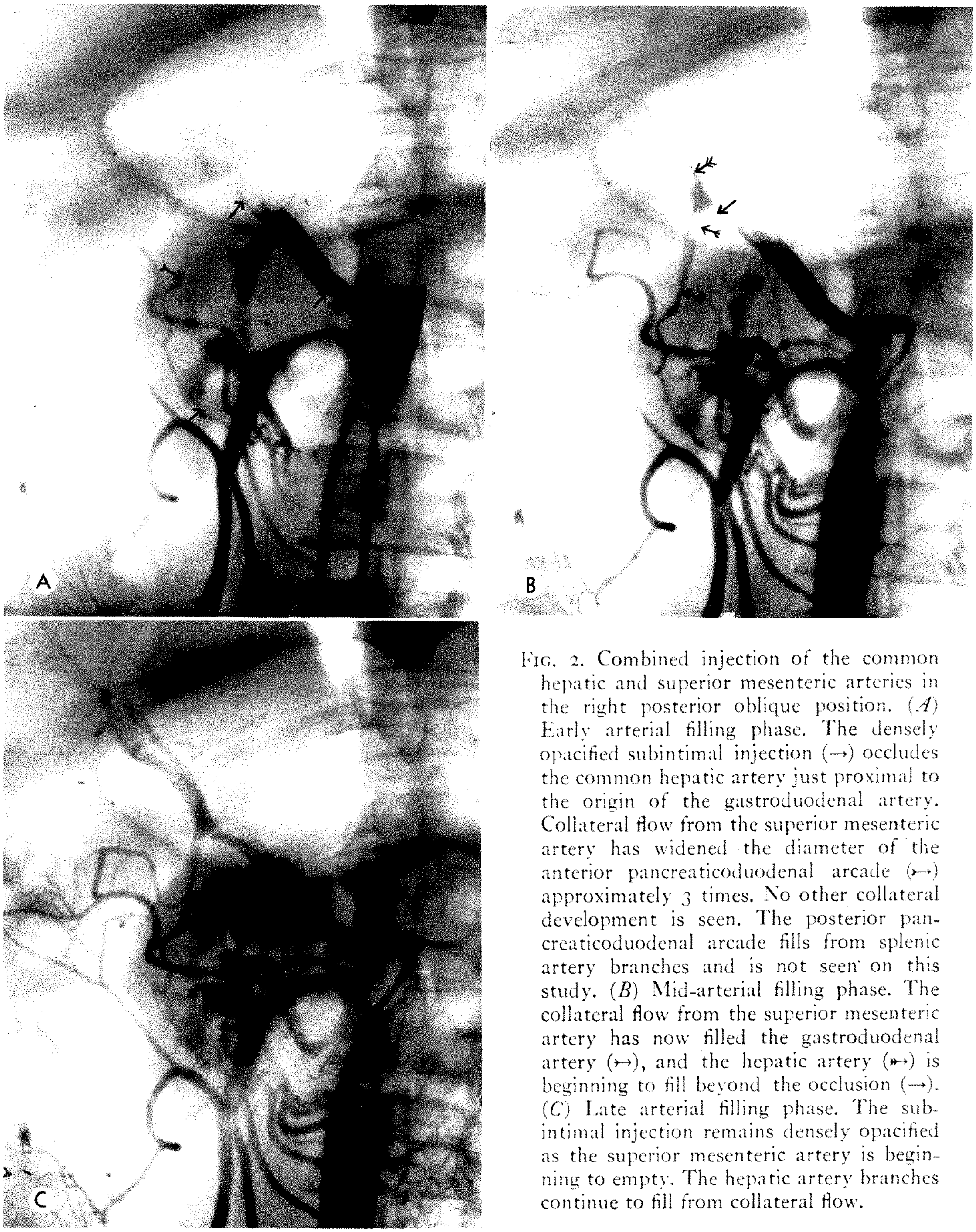

Fig. 2. Combined injection of the common hepatic and superior mesenteric arteries in the right posterior oblique position. (*A*) Early arterial filling phase. The densely opacified subintimal injection (→) occludes the common hepatic artery just proximal to the origin of the gastroduodenal artery. Collateral flow from the superior mesenteric artery has widened the diameter of the anterior pancreaticoduodenal arcade (↣) approximately 3 times. No other collateral development is seen. The posterior pancreaticoduodenal arcade fills from splenic artery branches and is not seen on this study. (*B*) Mid-arterial filling phase. The collateral flow from the superior mesenteric artery has now filled the gastroduodenal artery (↣), and the hepatic artery (⤞) is beginning to fill beyond the occlusion (→). (*C*) Late arterial filling phase. The subintimal injection remains densely opacified as the superior mesenteric artery is beginning to empty. The hepatic artery branches continue to fill from collateral flow.

and left gastro-epiploic arteries. These vessels could not be identified on any of the angiograms.

The dependence of 5 of the 7 major collateral pathways to the liver on the gastroduodenal artery helps explain the high mortality associated with sudden occlusion of the hepatic artery distal to this vessel,[6] compared with the absence of complications associated with proximal ligation.[1] That this patient had no symptoms during or after the occlusion would indicate the

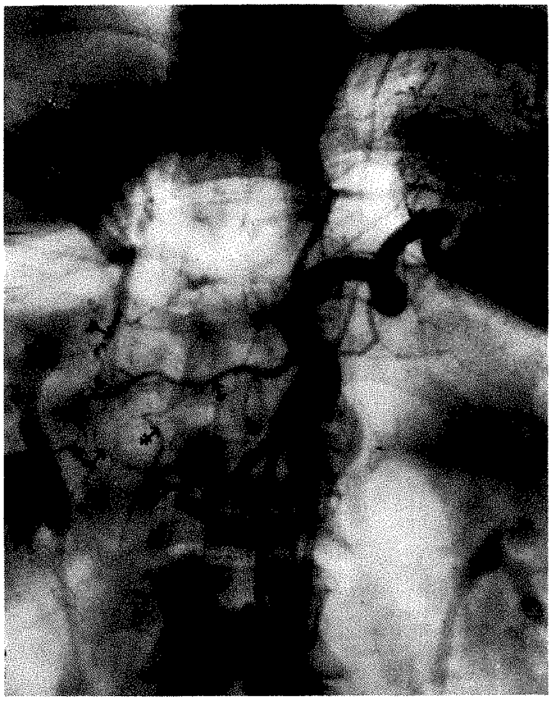

FIG. 3. Selective injection of the splenic artery (arising separately from the aorta) in the anteroposterior position. The retroduodenal branch of the dorsal pancreatic artery (↦) and its branch to the uncinate process (⇻) are enlarged and supply collateral flow to the liver via the posterior pancreaticoduodenal arcade (↣) and gastroduodenal artery. The anterior pancreaticoduodenal arcade, filling from the superior mesenteric artery, is not seen in this study.

adequacy of the angiograpically demonstrated collateral blood supply from other visceral arteries over the pancreatic and gastroduodenal arteries. How long this obstruction to the hepatic blood flow remained is not certain, since no follow-up examination was performed. Experience in other arteries has shown that 24 hours after a subintimal injection, the vessel will have an angiographically normal appearance.

Only 4 subintimal injections have occurred in this department in 800 selective celiac and superior mesenteric angiographies. This is probably because only end-open catheters are used for selective examinations. No injection is performed unless good backflow of blood from the catheter is first obtained. Such backflow indicates that the tip of the catheter is not against the wall of the vessel. If side holes are used, good backflow may be obtained even if the tip is occluded by its position against the wall. It is not certain why subintimal injection occurred in this patient, but it is probably secondary to recoil of the catheter, directing the jet against the vessel wall.

SUMMARY

An acute occlusion of the common hepatic artery by a subintimal injection of contrast medium has provided an opportunity to observe the immediate collateral blood flow to the liver. The vessels participating in the collateral supply were the dorsal pancreatic artery and its branches in the head of the pancreas, the pancreaticoduodenal arcades and the gastroduodenal artery.

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PORTAL VENOGRAPHY BY SELECTIVE ARTERIAL CATHETERIZATION*

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VISUALIZATION of the portal vein is important in the study of patients with portal hypertension both before and after surgical portal systemic shunts. Opacification of the vein can be accomplished by direct puncture of the splenic pulp with injection of a contrast agent, or by injection into the selectively catheterized celiac, splenic, or superior mesenteric artery. The contrast material injected arterially travels to the spleen or bowel and then returns by way of the splenic or intestinal veins, thus demonstrating the portal vein.

Portal vein opacification by the standard direct splenic pulp puncture technique was introduced in 1951 by Abeatici and Campi.[1] Ödman,[9] reporting his experience with selective catheterization of the celiac axis by the Seldinger[12] technique in 1958, first noted that contrast material injected by that route occasionally produced adequate opacification of the portal venous structures. Boijsen *et al.*,[3] in 1963, reported a series of cases of portal hypertension studied by selective celiac and superior mesenteric injection. In a number of these cases, he and his co-workers utilized simultaneous injection into the celiac and superior mesenteric artery in an effort to demonstrate the portal system more completely and improve concentration of the iodinated contrast material in the portal vein.

The present authors[10] in 1964 reported their experience with the arterial technique and described a new method whereby improved visualization of the portal venous system is achieved by injection into the selectively catheterized splenic artery. Selective splenic artery injection was an outgrowth of the observation that when an injection is made into the celiac axis in a patient in whom the hepatic artery arises from the superior mesenteric artery, as is the case in approximately 15 per cent of patients, or when selective splenic injections are performed for other indications, the splenoportogram is of better quality than in most instances of celiac injection. The purpose of this paper is further evaluation of this technique in 46 patients with suspected portal hypertension or thrombosis of the portal vein, or for confirmation of patency of a previous shunt. These cases are drawn from a series of 206 consecutive patients in whom selective celiac, splenic, hepatic, and/or superior mesenteric angiography was performed at the Massachusetts General Hospital.

TECHNIQUE

Materials and Methods. A percutaneous puncture is accomplished with a New[8] needle, and a preformed Kifa red catheter is inserted using the Seldinger technique. The puncture is generally performed via the femoral artery, although occasionally the left brachial or axillary route is used. When catheterization of the splenic artery is desired, the tip of the catheter may require accentuated or additional curves to aid in its advancement from the celiac axis into the splenic artery. Catheter introduction into the splenic artery has also been facilitated by the use of special curved guide wires with flexible tips. The catheterization is monitored with television fluoroscopy,

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TABLE I

INDICATIONS FOR SELECTIVE ARTERIAL CATHETERIZATION

Indications	Selective		
	Splenic	Celiac	Superior Mesenteric
Portal Hypertension			
Cirrhosis. ? Patent Portal Vein	15	9	1
Cirrhosis. ? Patent Portocaval Shunt	4		
After Splenectomy (Portal Hypertension)			
Cirrhosis. ? Patent Portal Vein			6
Cirrhosis. ? Patent Portocaval Shunt			1
Cirrhosis. ? Patent Splenorenal Shunt			1
Other			
Budd-Chiari Syndrome	2		
? Patent Portal Vein with Mass in Liver		3	1
? Patent or Invaded Vein with Pancreatic Mass	1		
Portal Hypertension with Arteriovenous Fistula		1	
Idiopathic Portal Hypertension	1		

and films are taken on a serial film changer, usually at a rate of one film every third second.

Large doses of contrast agent have been found necessary to give consistent opacification. Usually, at least 50 or as much as 75 cc. of 76 per cent renografin is injected into the splenic or superior mesenteric arteries. On the rare occasion when contrast material is dilute and the vein overlies the spine, the subtraction technique of Ziedses des Plantes[14] has been found helpful. With the use of subtraction, the bone density of the spine can be removed, thus showing the portal vein without the confusing overlying bone image. Since the routine use of larger doses of contrast material (50 cc. or more) has been adopted, however, subtraction has not been necessary.

RESULTS

Table I lists the indications for selective splenic, celiac, and superior mesenteric angiography in the 46 patients in whom visualization of the portal venous system was desired. In addition to these 46 cases, portograms were obtained incidentally in many of the other 160 patients in whom celiac, superior mesenteric, and selective splenic injections were performed for other indications. Only in three-fourths of 84 cases of celiac axis injection with films exposed at adequate intervals was there satisfactory visualization of the portal vein. Successful selective splenic injection yielded good splenoportograms in over 90 per cent of cases. In all instances in which both celiac axis and selective splenic artery studies were obtained, the splenoportogram after selective splenic artery injection was of better quality than after celiac axis injection (Fig. 1, *A–D*).

Table II lists the various catheterization procedures attempted primarily for portal venous visualization and shows the incidence of success and failure of specific vessel catheterization as well as the incidence of satisfactory portal venous visualization. Selective splenic artery catheterization was accomplished in 17 of 23 cases in which it was attempted (Fig. 2, *A* and *B*; and 3, *A* and *B*). In all but 2 of the 17, visualization of the portal vein was adequate. In 1, the catheter slipped out during injection and because of poor patient cooperation, the injection was not repeated. In the other, a patient with a previous end-to-side portocaval shunt, there was no visualization of

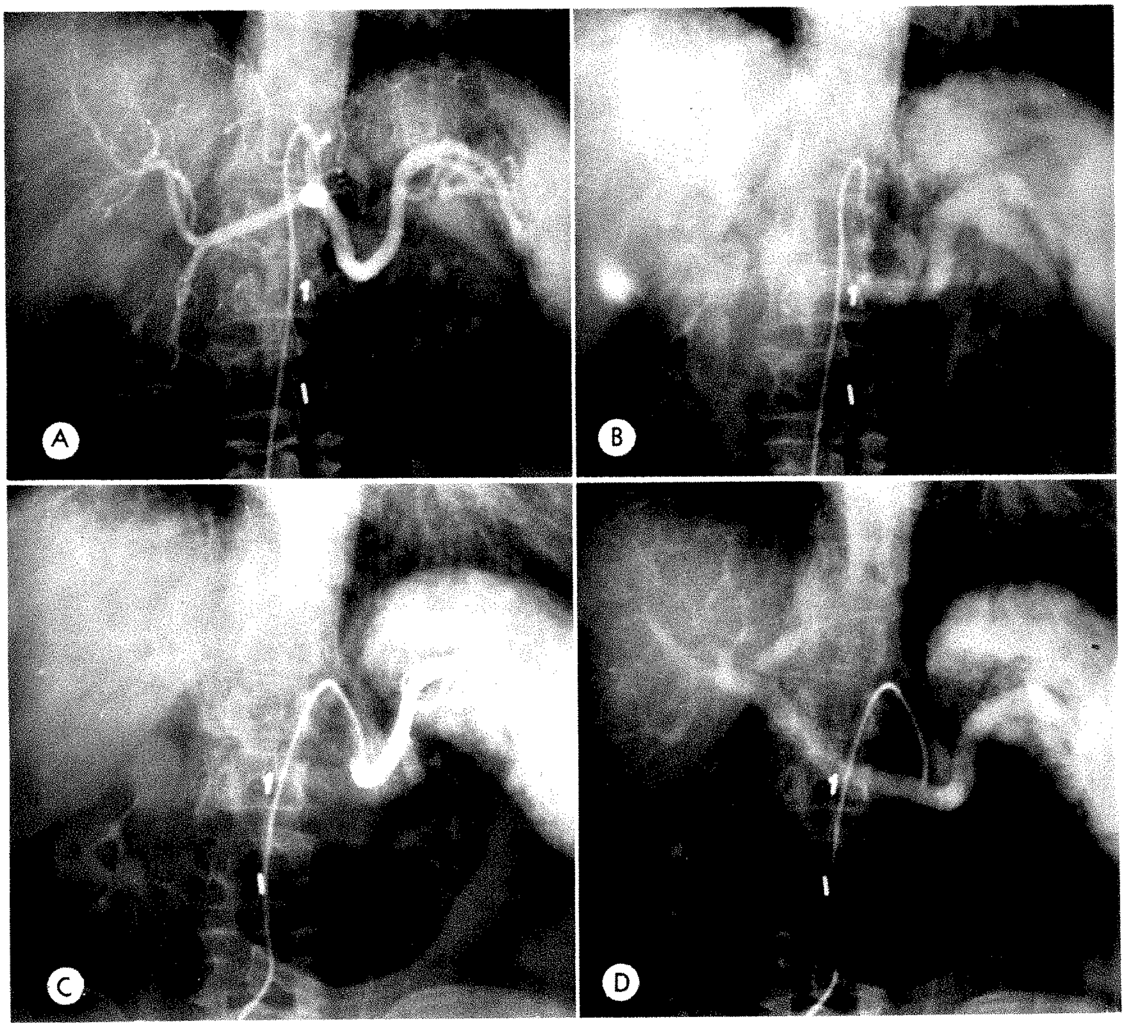

Fig. 1. Advantage of selective splenic artery over celiac axis catheterization in a normal patient. (*A*) Selective celiac arterial injection. (*B*) Portal venogram from celiac axis injection. (*C*) Selective splenic arterial injection. (*D*) Improved portal venous opacification from selective splenic injection.

Table II

TECHNICAL RESULTS OF SELECTIVE ARTERIAL CATHETERIZATION FOR PORTAL VENOUS EVALUATION (PRIMARY EXAMINATION)

Type of Catheterization	Total Cases	Successful Arterial Catheterization	Adequate Venous Visualization	Failure of Catheterization	Failure of Visualization*
Selective Splenic	23	17	15	6†	4
Selective Celiac	13	12	10	1	3
Selective Superior Mesenteric	10	10	10		0
All	46				7

* See also text.

† Adequate visualization obtained with celiac catheterization in 1; with superior mesenteric catheterization in 3.

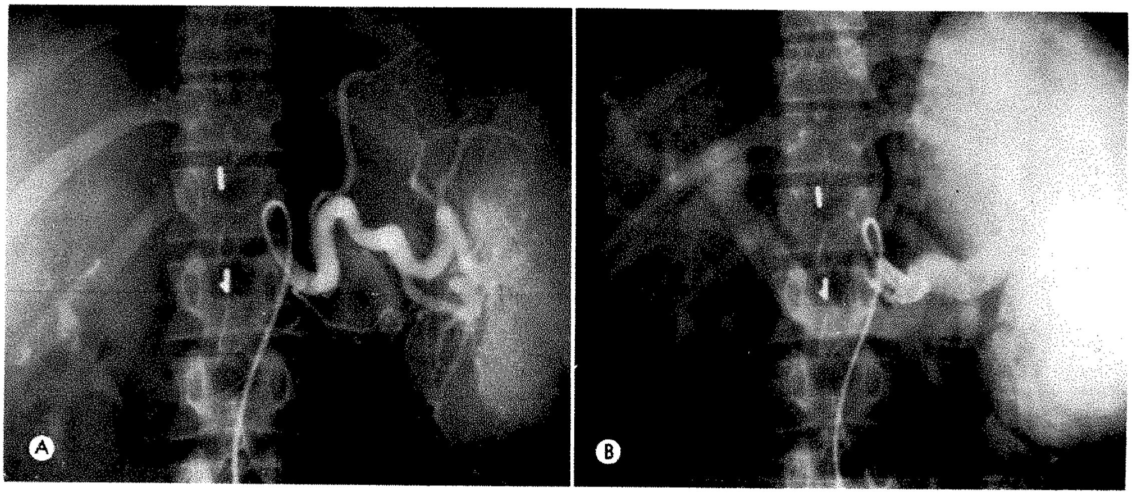

FIG. 2. Patent portal vein and collaterals in cirrhosis with portal hypertension and bleeding varices. (*A*) Selective splenic arterial injection. (*B*) Portal venogram showing the portal vein to be patent, with retrograde flow through inferior mesenteric and coronary veins with filling of gastric and esophageal varices. (Intramural deposition of contrast material in splenic artery caused no symptoms.)

the shunt, the splenic, or collateral veins despite a good injection. A double catheterization of both celiac and superior mesenteric arteries was attempted, but this also failed to demonstrate the portal venous system. Since no proof was available, this attempt was classified as a failure to visualize, although thrombosis of an end-to-side portocaval shunt was clinically suspected.

In the 6 patients in whom catheterization of the splenic artery was unsuccessful, adequate splenoportograms were obtained from celiac or superior mesenteric cathe-

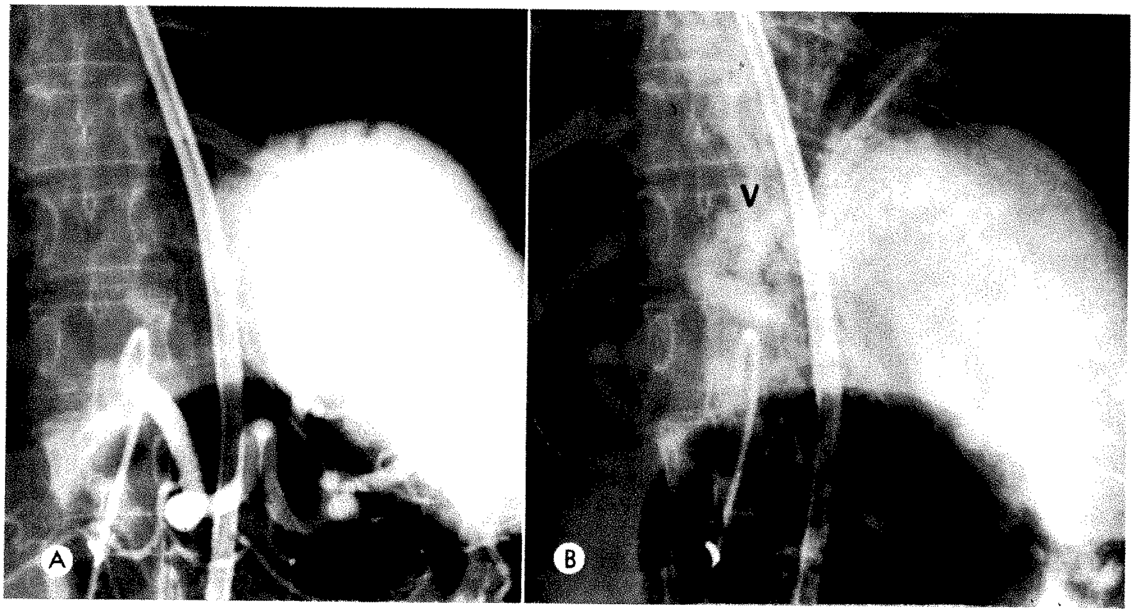

FIG. 3. Ineffective but open portocaval shunt in a patient with cirrhosis (proved by autopsy). (*A*) Selective splenic arterial injection. (*B*) Venogram showing shunt to be patent, with a small amount of contrast material passing through the shunt (S) into inferior vena cava (IVC) but flow predominantly passing through collaterals and varices (V).

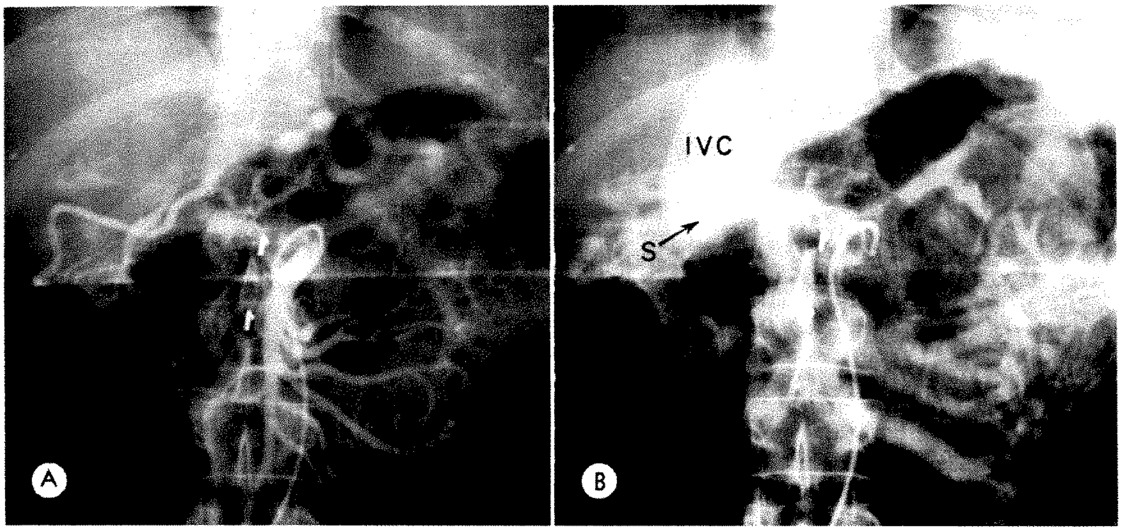

FIG. 4. Patent portocaval shunt in cirrhotic male. (*A*) Selective superior mesenteric artery injection. (*B*) Superior mesenteric vein is opacified and shows flow through a patent shunt (S) into inferior vena cava (IVC).

terization in 4. In 1 of the 2 remaining failures, catheterization of any vessel supplying the upper abdominal viscera was prohibited by iliac artery atherosclerosis; in the other failure, catheterization of the splenic artery was difficult and further attempts to enter this vessel or the celiac or superior mesenteric artery had to be terminated.

In 13 patients in whom celiac catheterizations were primarily attempted for the diagnosis of portal hypertension (early in the series), there was 1 failure because of inability to advance the catheter beyond the iliac vessels due to atherosclerosis. In 2 of the 12 cases in which the vessel was successfully catheterized, the portal vein was not adequately demonstrated, and attempts at catheterization of other vessels were not made as the value of selective splenic or superior mesenteric artery catheterization was not appreciated at that time.

Of the 10 attempts at primary superior mesenteric catheterization, all were successful, with adequate visualization of the portal venous system (Fig. 4 through 7).

In addition to the above 46 cases, 4 selective splenic artery catheterizations were performed for indications other than portal venous visualization, and in all excellent splenoportograms were obtained. In these splenoportograms, opacification of the portal vein was superior to that following celiac injection. The portogram from superior mesenteric artery injection with high doses of contrast material was often comparable to that obtained with selective splenic artery injection.

Over-all satisfactory visualization of the portal veins by arterial injection occurred in 39 out of 46 cases, or 85 per cent. Had further attempts been made at selective catheterization, the incidence of visualization could almost certainly have been improved. Esophageal varices are not invariably shown by this technique, but evidence of collateral retrograde flow into the coronary and inferior mesenteric veins is almost always seen in portal hypertension.

Of the 19 cases subsequently verified by surgery or autopsy, the angiographic interpretation was correct in all but 1. In this one instance the angiogram was interpreted as showing a patent but slightly narrowed portal vein. In actual fact the vein had been severely thrombosed, although a small portion of the lumen was unobstructed. This must be regarded as an error of inter-

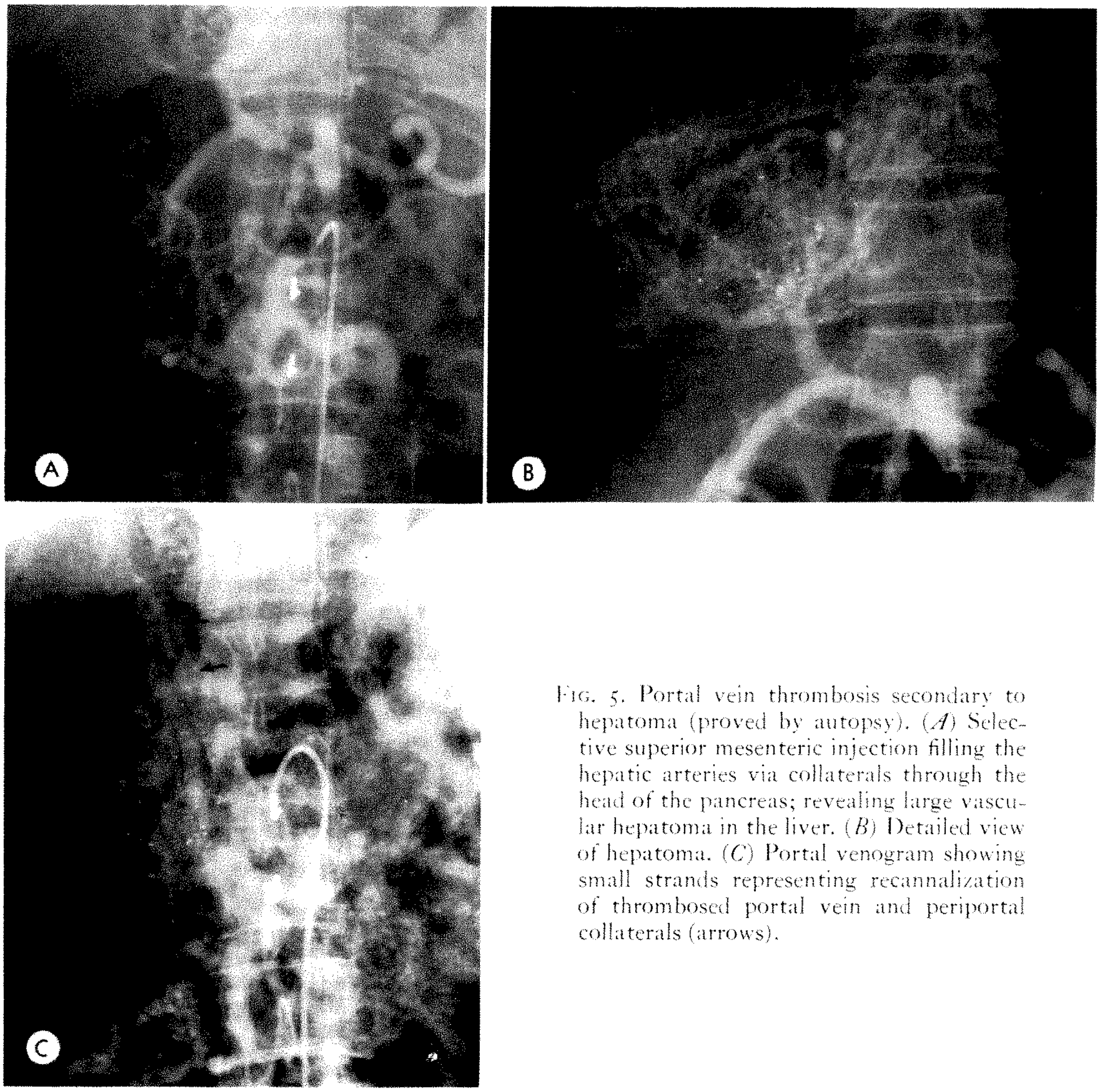

Fig. 5. Portal vein thrombosis secondary to hepatoma (proved by autopsy). (*A*) Selective superior mesenteric injection filling the hepatic arteries via collaterals through the head of the pancreas; revealing large vascular hepatoma in the liver. (*B*) Detailed view of hepatoma. (*C*) Portal venogram showing small strands representing recannalization of thrombosed portal vein and periportal collaterals (arrows).

pretation rather than a deficiency in the angiogram.

The complications of the Seldinger technique have been previously described.[5] In this series of 46 patients, 1 had temporary loss of a dorsalis pedis pulse. In one instance there was extravasation of a small amount of contrast material subintimally into a selectively catheterized splenic artery without reduction in flow or any symptomatology (Fig. 2, *A* and *B*). Rarely, a patient was mildly nauseated for as long as 24 hours, and, occasionally, low-grade fever was present for 24 hours without obvious cause. There was no loss of use of limb, no significant hematoma formation at the puncture site, nor any known significant abdominal vascular or visceral complication.

DISCUSSION

The visualization of the portal venous system is important for determining whether portal hypertension is due to intrahepatic block secondary to cirrhosis or to an extrahepatic block caused by portal vein thrombosis. It is valuable for demonstrating patency of the portal vein prior

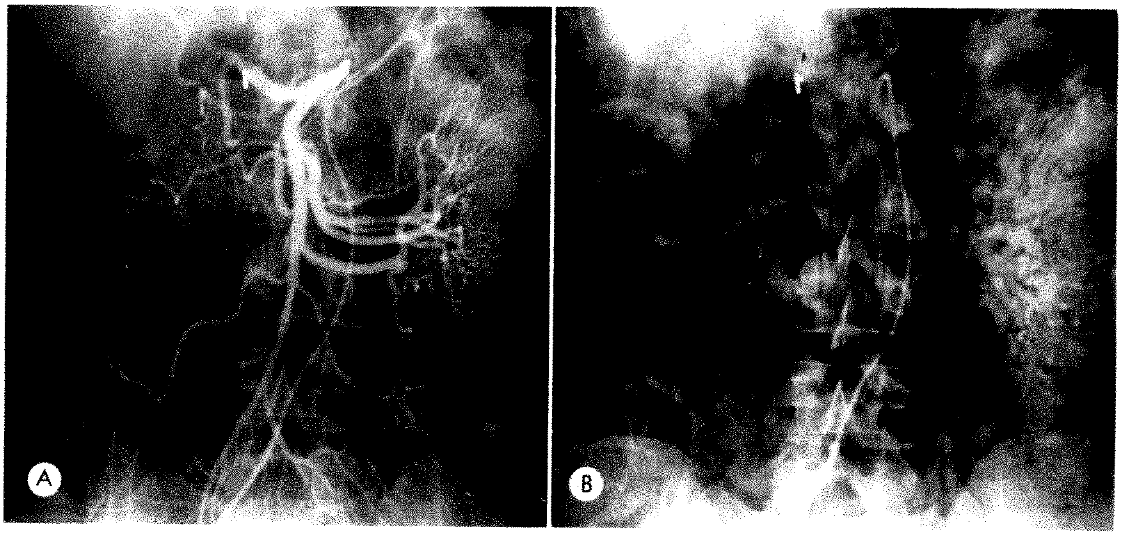

Fig. 6. Portal vein thrombosis since childhood in a female with otherwise normal liver and previous splenectomy (thrombosis surgically confirmed). (*A*) Selective superior mesenteric artery injection. (*B*) Portal venogram showing mesenteric varices and multiple collaterals to the liver with channels through the gallbladder bed.

to performance of a portocaval or splenorenal shunt to decompress the portal system (Fig. 2, *A* and *B*). In the event of portal vein thrombosis, the procedure may demonstrate large intestinal veins or other collateral channels (Fig. 6*B*), which, if of sufficient caliber, may be utilized for shunts. In upper gastrointestinal bleeding confirmation of esophageal varices has been obtained. Postoperatively, the patency of shunts has been demonstrated (Fig. 3, *A* and *B*; 4, *A* and *B*; and 7, *A*, *B* and *C*). Splenoportography has also been used to demonstrate invasion of the splenic or portal veins by pancreatic or other neoplasms. Masses within the liver may on occasion be detected.

The direct percutaneous splenic pulp puncture is still a good method for splenoportography because of the denser opacification of the portal vein thus obtained and in view of the simplicity of the technique. It also has the advantage of providing for measurement of the portal pressure as reflected by the splenic pulp pressure. This method, however, should be used only with fairly rigid criteria to prevent post-puncture bleeding. Patients must be carefully screened to rule out a low prothrombin time, not infrequently found in cirrhosis, or a decreased platelet count, which may be associated with hypersplenism secondary to cirrhosis. Patients with massive ascites are also poor candidates for direct splenic pulp puncture, since the tamponading effect of the spleen against the abdominal wall is lost with the displacement of the organ by ascitic fluid. Again, when splenomegaly is due to infection, the spleen is unduly friable and should not be subjected to direct puncture. In the normal nonfibrotic spleen, direct percutaneous puncture may be attended by undue risk and is technically more difficult because the spleen is small. Hazard is increased with multiple punctures. Mild bleeding is common following puncture splenoportography, and severe loss of blood requiring transfusions, as well as splenic rupture and death, has been reported.[2]

Arterial injection of contrast material seems a more physiologic method of demonstrating the portal circulation and by this means many of the objections to direct puncture are obviated. Arterial portal venography can be safely accomplished in most instances in which direct puncture is contraindicated. Although severe bleeding

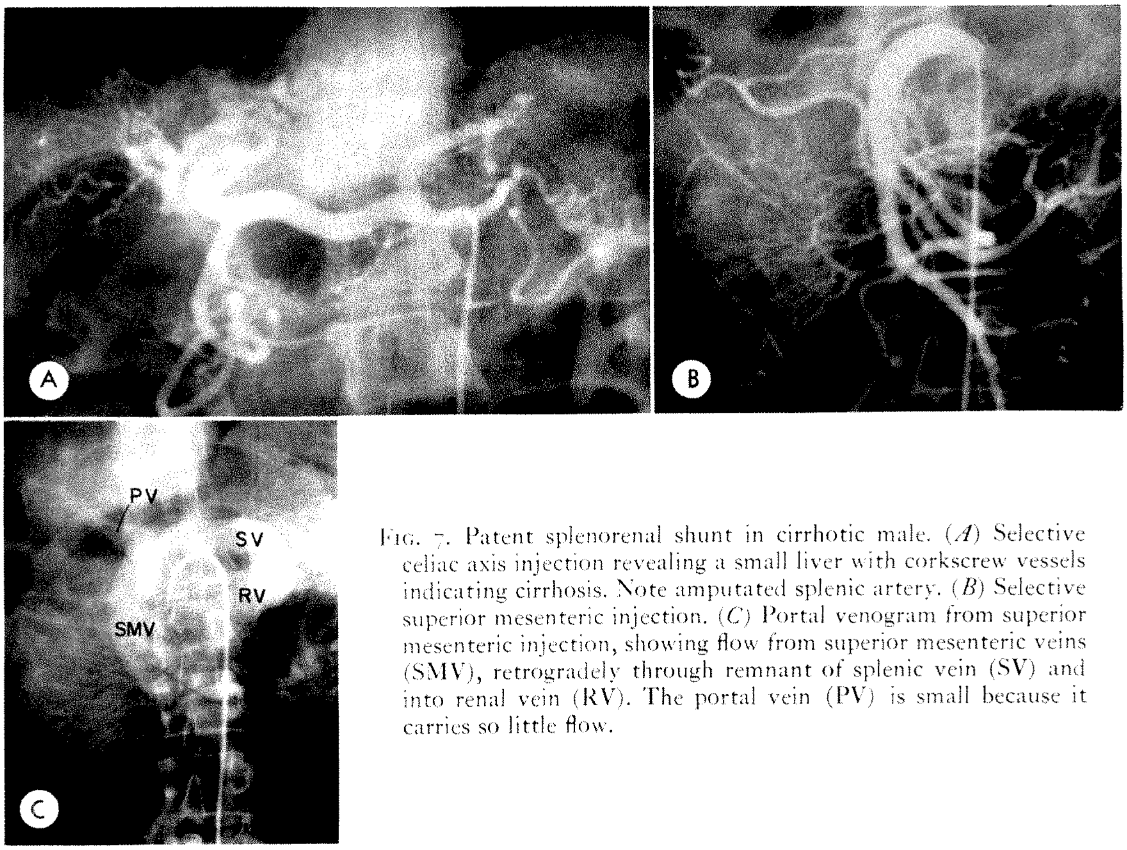

FIG. 7. Patent splenorenal shunt in cirrhotic male. (*A*) Selective celiac axis injection revealing a small liver with corkscrew vessels indicating cirrhosis. Note amputated splenic artery. (*B*) Selective superior mesenteric injection. (*C*) Portal venogram from superior mesenteric injection, showing flow from superior mesenteric veins (SMV), retrogradely through remnant of splenic vein (SV) and into renal vein (RV). The portal vein (PV) is small because it carries so little flow.

diatheses causing purpura and spontaneous bleeding from mucous membranes, etc., are still to be regarded as contraindicating arterial catheterization, the mild depression of prothrombin time and platelet count seen in cirrhosis with portal hypertension does not prohibit its use. In patients with prothrombin times as low as 20 per cent, platelet counts of 50,000, or ascites, the procedure was performed safely, without any serious complication or sequelae. Particularly good splenoportograms are obtained with small normal spleens because contrast material is less diluted. It has also been performed without complication in patients in whom previous attempts at splenic pulp puncture were unsuccessful and should be safe in patients with infective splenomegaly. Another advantage of arterial injection is the possibility of performing multiple injections in different projections with safety, whereas it is generally advised that only one direct injection be made into the splenic pulp.

Occasionally after direct puncture only collateral vessels fill with contrast medium, either because of portal vein thrombosis or as a result of a functional derangement secondary to portal hypertention with increased resistance in the portal hepatic bed and collateral flow. In these instances arterial injection can be performed into both celiac and superior mesenteric systems—or, even better, combined selective splenic and superior mesenteric injection—producing opacification of practically the entire venous inflow and demonstrating the portal vein if it is patent and flow is in the normal direction. It is possible, however, for retrograde flow to occur through the portal vein either due to cirrhosis or hepatic vein thrombosis. In cirrhosis there may be a postsinusoidal outflow obstruction,[13] while in hepatic vein thrombosis there is usually a total block of hepatic venous egress. In these instances the blood flows from the hepatic arteries through the sinusoids into the portal venules and retrogradely into the portal vein, due to obstruction in the hepatic venous outflow, and a false impression of portal vein thrombosis may be obtained. In 5 cases of cirrhosis in which selective hepatic angiography was performed, however, no retrograde flow into the portal vein was observed.

In the splenectomized patient, visualization of the portal vein is manifestly impossible by direct splenic puncture. Demonstration of the vessel in the presence of bleeding under these circumstances is of importance because of the possibility of portal vein thrombosis, and the information thus obtained is essential prior to contemplated surgical shunting procedures. Several techniques have been proposed in the past for preoperative visualization of the portal vein in patients who have had splenectomies, but all have proved impractical or have yielded inconsistent results. Such techniques include operative cannulation of hemorrhoidal veins with injection into the inferior mesenteric system and cannulation of the umbilical vein in the hope of defining the portal vein. The former method requires general anesthesia and the latter is not always possible because the umbilical vein may not be patent. In both procedures injection may be against the direction of blood flow in a patient with portal hypertension and results may thus be unreliable. Percutaneous transhepatic injection into the portal vein or high pressure injections into the hepatic vein, with retrograde transhepatic flow into the portal systems, may be hazardous and do not adequately fill the portal and splenic veins through their entire length. Injection into a superior mesenteric venous tributary at laparotomy has been used but does not allow preoperative evaluation; often film quality is poor, and serial splenoportograms cannot usually be obtained in the operating room. Catheterization of the superior mesenteric artery is the only practical preoperative means of producing consistently diagnostic splenoportograms when the spleen has been removed. It is more reliable, safer, and easier of performance than the other methods.

Superior mesenteric artery catheterization is also useful to confirm patency of previous shunts in patients who have undergone splenectomy. Portocaval shunt patency may, however, be demonstrated also by direct catheterization of the shunt from the inferior vena cava or, if the spleen has not been removed, by conventional puncture splenoportography or selective splenic arterial injection.

A splenoportogram obtained by arterial injection has the advantage that specific information about the liver is provided when the tip of the catheter is placed in the hepatic or celiac artery and a hepatic angiogram is obtained. With the hepatic angiogram, confirmation of cirrhosis or suspected hepatoma with secondary thrombosis of the portal vein as a cause of portal hypertension is usually possible[7,11] (Fig. 5, *A*, *B* and *C*; and 7, *A*, *B* and *C*). Although liver masses can be shown as filling defects in the hepatogram phase of a direct puncture splenoportogram if the portal vein is patent, their nature cannot be ascertained. Celiac or hepatic arteriography, on the other hand, will often demonstrate the tumor vascularity pathognomonic of malignant neoplasia since most malignant tumors of the liver, either primary or metastatic, derive their blood supply from the arterial circulation.[4]

Arterial portal venography produces less contrast density in the portal vein than direct splenic pulp puncture, due to dilution in the splanchnic bed, probably because water is drawn in by the high osmotic concentration of the contrast agent while it is in the capillaries. Direct splenic puncture deposits the injected medium directly into the veins or close to them, thus increasing contrast density.

Aortic injection is generally inadequate to produce a splenoportogram; the contrast material is diluted in the aorta and too little arrives in the spleen. Selective celiac axis injection is a great improvement over an aortic injection, but still much of the medium escapes into the hepatic and gastric arteries. For a given volume of contrast material, a selective splenic artery injection delivers the most concentrated and largest bolus of the agent into the spleen, thus improving the density of the splenoportogram (Fig. 1*D*). Generally, also of the arterial methods, selective splenic artery injection produces the best quality venogram, although selective superior mesenteric artery injection is often comparable. If selective splenic artery or celiac axis injection is impossible, a superior mesenteric artery injection will almost always give the necessary information, though larger doses are frequently required to obtain a result comparable to a selective splenic artery injection. The superior mesenteric arterial route of administration, of course, will not demonstrate the splenic vein.

Another advantage of selective splenic artery injection over the celiac axis injection is that filling of the left gastric artery is avoided, and thus the gastric bed, which is drained by the coronary vein, does not receive contrast material. The coronary vein, therefore, will not be opacified unless portal hypertension with retrograde flow is present (Fig. 2, *A* and *B*). With celiac axis injection, contrast material fills the left gastric artery and in that instance filling of the coronary vein cannot be considered abnormal; thus one criterion for judging portal hypertension is invalidated. Inferior mesenteric venous filling in retrograde fashion is valid evidence for portal hypertension under most circumstances of arterial injection, be it celiac, selective splenic, or superior mesenteric.

A large bolus (50 to 75 cc.) of 76 per cent renografin is essential to obtain consistent results by either splenic or superior mesenteric artery injection. Particularly are the larger doses essential when the spleen is enlarged or when there is portal hypertension with mesenteric varices. With a small spleen of normal size, 20 to 30 cc. is sufficient. The doses used have not been attended by any overt complication and the bowel has not shown any gross abnormalities at operations performed from a few days to months later. Some patients have experienced nausea for as long as 24 hours, but this has always subsided without further discomfort. Since the ultimate effects of large doses of currently available contrast agents (renografin in this series) on the human bowel are not known, the selective splenic arterial route of administration seems preferable.

The use of vasodilator and vasospastic drugs to alter flow patterns needs further investigation. Kahn and Callow[6] have noted enhancement of portal venous opacification with the use of these agents, and preliminary experience in this series agrees with their observation.

SUMMARY

Arterial methods for visualization of the portal venous system are a practical alternative to conventional direct splenic pulp puncture. Arterial injections are applicable when splenic pulp puncture is contraindicated and consistently produce good opacification. Arterial portography can be performed even in the presence of mild bleeding states, when the spleen is normal or small in size, in infective splenomegaly, when a previous attempt at splenic pulp puncture has been unsuccessful, when a splenoportogram reveals filling only of collaterals, and in patients who have previously undergone splenectomy.

Selective catheterization with injection of the splenic artery is the preferable arterial technique but, if catheterization of this vessel is unsuccessful, selective superior mesenteric artery injection usually produces comparable results. When the spleen has been previously removed, selective superior mesenteric artery injection is the only practical method of showing the portal venous system preoperatively.

Adequate visualization of the portal system was obtained in 85 per cent of 46 patients in whom the arterial technique was used for suspected portal hypertension or

thrombosis of the portal vein, or for confirmation of the patency of a previous shunt.

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ARTERIAL STATIONARY WAVES*

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DURING the past few years, there have been occasional reports concerning the visualization on arteriograms of an unusual arterial deformity—so-called stationary waves. To the best of our knowledge, no corresponding pathologic changes have been observed to explain the nature of this finding, which has, therefore, been the subject of considerable speculation. The purpose of the author is to review the available literature on the subject and to present some evidence concerning the etiology of this arteriographic appearance.

REVIEW OF LITERATURE

In 1960, Theander[11] described a peculiar arteriographic appearance in large, medium-sized and small arteries in the lower extremities in 4 patients, from a study of a series of 217 consecutive lower extremity angiograms in 160 patients. In each case, the involved arterial segment exhibited a very regular series of ripple-like deformities or corrugations of lumen, resembling a string of flattened beads. He noted that the waves occurred proximal to some obstructing vascular lesion in each case. In 1 patient embolization occurred 6 hours before angiography was carried out; in another, 4 months previous to angiography; 1 patient was diagnosed as having Buerger's disease and 1 patient had a vascular disease of obscure origin.

Wickbom and Bartley[12] had previously reported a study of arterial spasm based upon 108 upper and lower extremity angiograms performed by percutaneous catheterization. They noted that the incidence of spasm in the peripheral arteries tended to be inversely proportional to age and illustrated several examples of irregular arterial narrowing, which disappeared following the intra-arterial injection of priscol. One of their illustrated cases appeared unique in that the involved femoral artery showed a series of strikingly *regular* corrugations with smoothly alternating areas of narrowing and relative widening. The patient was 32 years of age and was diagnosed as having Buerger's disease. The study was repeated 3 weeks later, at which time these corrugations were even more striking and involved the same segment of the femoral artery over a distance of several centimeters. At the same time, the deep femoral artery showed a severe smooth spastic narrowing over a moderate distance. Following injection of priscol through the catheter, the corrugations diminished, but now the whole vessel was smoothly narrowed.

Sutton,[7] in 1962, reported observing this type of arterial deformity in about one dozen femoral arteriograms, approximately one-third of which were in patients diagnosed as having Buerger's disease. He stated that the phenomenon is extremely rare in other vessels, but that he has noted this appearance on occasion in the iliac arteries and cited single examples in the internal carotid and in the renal artery.[7,8,9]

Schechter,[6] discussing the incidence of spasm of the internal carotid arteries during percutaneous catheterization, mentioned the so-called stationary waves and illustrated purported examples.

Mayall[4] recently described arterial corrugations appearing in 2 branches of the superior mesenteric artery on aortograms. Mayall also reported a personal communication from Theander, in which Theander states that he has seen waves in several additional femoral arteriograms, all but one showing arterial block peripherally and that he had also seen examples of the phenomenon in the iliac arteries proximal to arterial compression used for pelvic arteriography by Fernström's method.

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The great rarity of the phenomenon of so-called stationary waves in the internal carotid artery and the finding of one clear-cut example in this vessel prompted a detail review of some 2,000 carotid angiograms and a consideration of the conflicting theories of origin of the phenomenon.

Most of the reviewed carotid angiograms were obtained by percutaneous puncture of the common or proximal internal carotid arteries with a needle cannula. In the large majority, the common carotid artery was punctured and the tip of the cannula lay in the distal common carotid artery or proximal internal carotid artery. In the 2,000 angiographic studies reviewed, 4 clear-cut examples of arteriograms (of 2 patients) showing arterial corrugations involving the cervical portions of the arteries and 2 angiograms (of 1 patient) showing rather limited involvement, similar to that illustrated by Sutton,[7] were discovered. (No examples of the phenomenon were found in the internal carotid arteries of patients in whom the carotid system was catheterized.)

REPORT OF CASES

Case i. J.R., a 28 year old male. Right carotid arteriography was performed several hours following acute subarachnoid hemorrhage. The patient had not lost consciousness but was grossly confused and disorientated. The spinal fluid pressure on the day of admission was over 500 mm. of water, with bright bloody fluid.

Quite well developed stationary waves were present over several centimeters of the cervical portion of the internal carotid artery. The carotid sinus was not involved (Fig. 1, *A* and *B*).

Left internal carotid angiography, on the next day showed a similar appearance of the internal carotid artery, again without involvement of the carotid sinus (Fig. 2, *A* and *B*).

Bilaterally, the carotid siphon gave rise to the posterior cerebral artery. On the left, there was also reflux into the basilar artery, with filling of both superior cerebellar arteries.

The posterior circulation was examined by bilateral injections, and these revealed no significant abnormality. No source of hemorrhage was found in this patient.

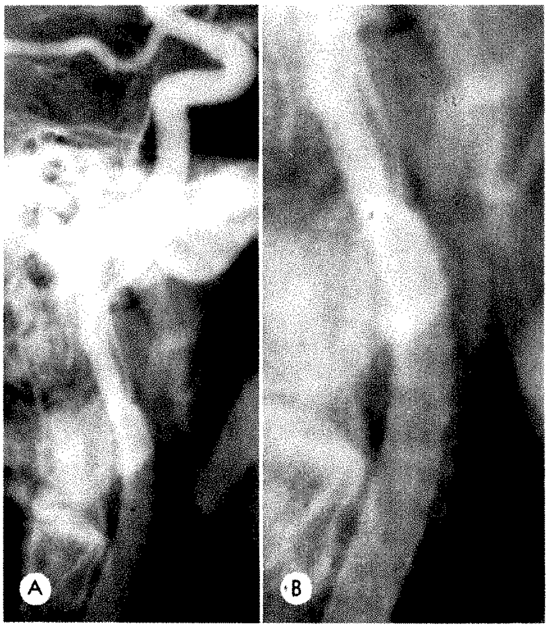

Fig. 1. Case i. (*A*) Right carotid angiogram. Lateral projection of initial arterial phase. The lumen of the internal carotid artery shows a smoothly corrugated outline. The main bolus of contrast medium has passed into the intracranial vessels and the more dilute contrast medium in the cervical portion of the artery has produced a "banded" appearance, with alternating transverse zones of increased and decreased density corresponding to the alternating bulged and constricted portions of the lumen. (*B*) Magnification of the involved segment of the internal carotid artery at the level of C-1 and C-2.

Case ii. H.R., a 20 year old male. Right carotid angiography was performed 10 days after a subacute subarachnoid hemorrhage. The common carotid artery was injected. Arterial waves were clearly visible in the internal carotid artery in both lateral and anteroposterior oblique projections (Fig. 3*A*). Some filling of the posterior cerebral artery occurred via the posterior communicating artery. There was spontaneous bilateral pericallosal arterial filling. A 6 mm. aneurysm of the right anterior cerebral-anterior communicating arterial junction with an apical bleb was demonstrated. There was no indication of spasm of the intracranial vessels. No lateral ventricular dilatation was evident. Left carotid angiography was per-

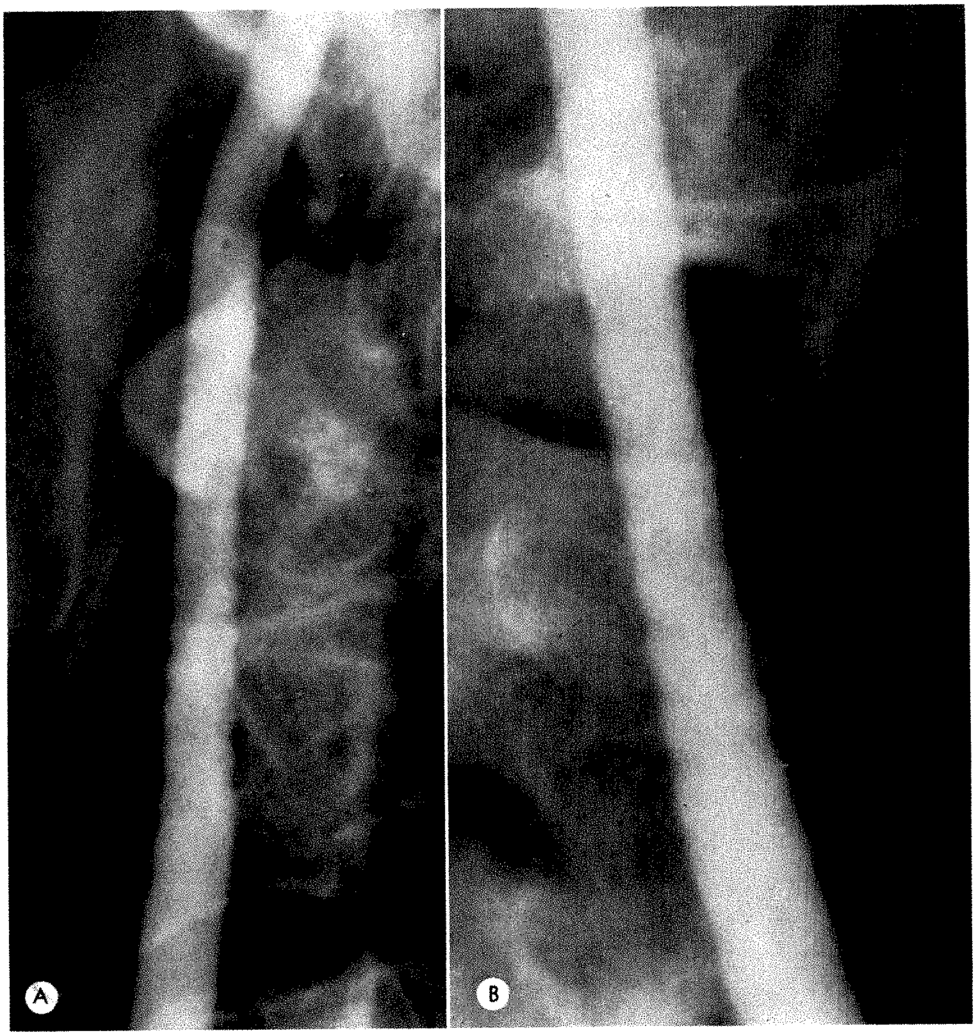

Fig. 2. Case I. (*A*) Left carotid angiogram showing the corrugated configuration of a portion of the cervical segment of the internal carotid artery. This portion of the vessel is filled with the main bolus of contrast material and horizontal banding is not as striking, although the corrugations are a little more pronounced than in the right internal carotid artery. (*B*) The involved segment of the internal carotid artery is shown in frontal projection, with the neck somewhat rotated to the right.

formed 2 days later. No arterial waves were demonstrated in the left carotid system and no additional aneurysms were found.

The patient did well following an operation in which the aneurysm was coated with Handa plastics. A routine postoperative right carotid angiogram with internal carotid injection was made 9 months later. Corrugation, with much the same appearance as before, was demonstrated in the internal carotid artery (Fig. 3, *B* and *C*). Again, there was no ventricular dilatation. The aneurysm was somewhat larger, but the apical loculus had become obliterated.

Case III. J.C., a 27 year old female. The patient presented with a subarachnoid hemorrhage. A right common carotid angiogram showed quite marked generalized contraction of the entire internal carotid artery (except for the carotid sinus). This was most severe in the distal siphon and its branches were markedly and smoothly narrowed. A posterior communicating artery infundibulum was demonstrated. There were also a few small arterial waves over a 1.0 to 1.5 cm. segment of the internal carotid artery, 2 cm. distal to the carotid sinus (Fig. 4*A*). A right carotid angio-

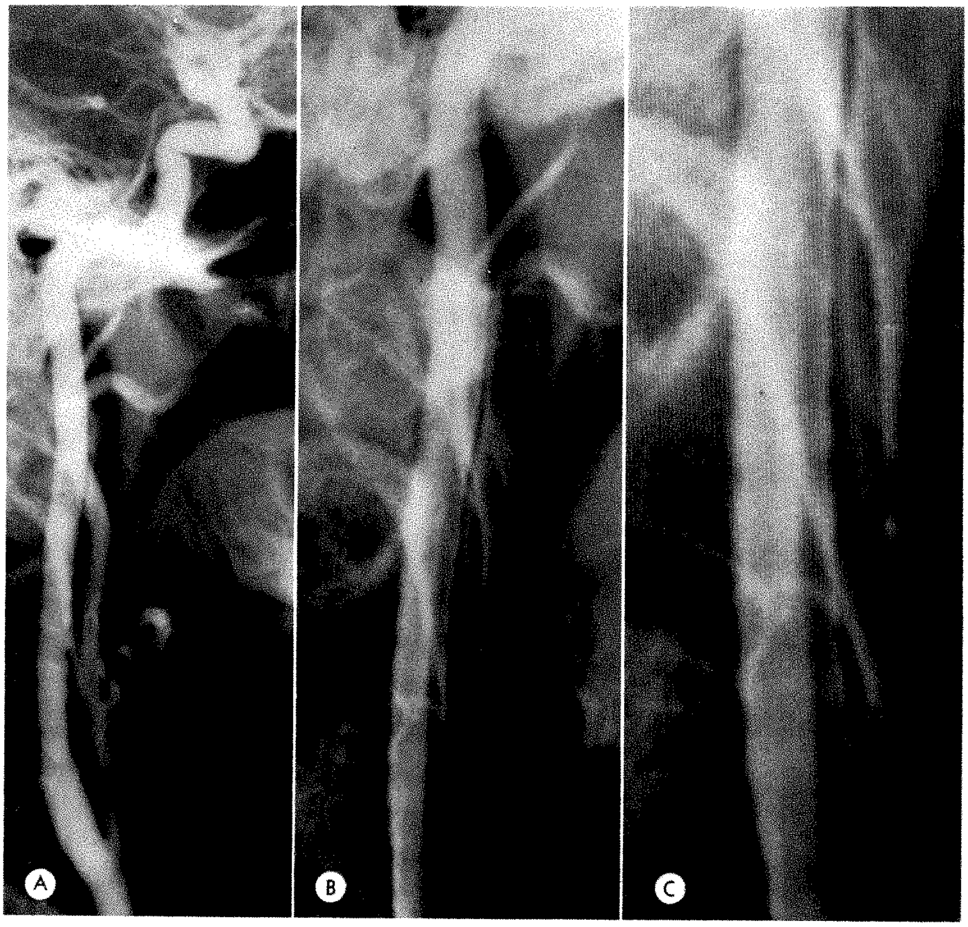

Fig. 3. Case ii. (*A*) Initial right carotid angiogram. The corrugated appearance of the proximal cervical segment of the internal carotid artery is visible. The carotid sinus is not involved. (*B*) Second right carotid angiogram showing again a corrugated appearance. The main contrast bolus has passed distally and horizontal "banding" is quite apparent. Note also that although some posterior layering of contrast material is present in the involved segment, the anterior wall of the artery is deformed to the same extent as the posterior wall. A very narrow layer of dense contrast material is shown clinging to the anterior wall of the artery, clearly demonstrating the cause of the appearance of horizontal banding when the main contrast bolus has passed distally. (*C*) Magnification of the involved segment shown in *B*.

gram by retrograde brachial catheterization 3 weeks later showed only slight residual narrowing of the internal carotid artery and no waves were visible (Fig. 4*B*). The vertebral arteries were of normal appearance.

No aneurysm was demonstrated in this patient and ventricular enlargement was not present. A left carotid angiogram showed a few very minor waves over a short segment of the internal carotid artery.

Comment. It is interesting that in Cases i and ii, the waves were also visible on roentgenograms of small low pressure test injections of contrast medium. In all 3 patients the carotid punctures were performed without difficulty. There were no intramural or perivascular injections.

Roentgenograms showing the phenomenon in other portions of the arterial tree have been collected and studied (Fig. 5 through 9). Previously published cases showing the phenomenon of stationary arterial waves and those from the present series are summarized in Table i.

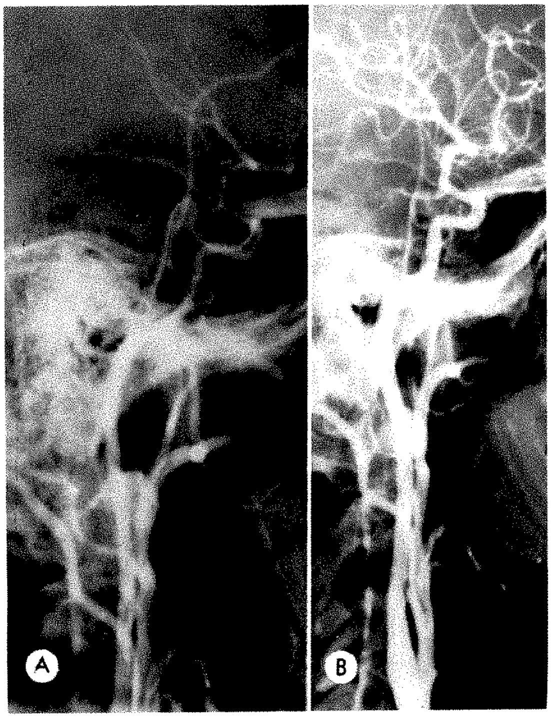

Fig. 4. Case iii. (*A*) First right carotid angiogram. Corrugations are visible in the internal carotid artery over a short segment at the level of C-2. The whole internal carotid artery is narrowed, particularly the more distal portions and there is striking narrowing of the intracranial branches. An infundibulum is present at the origin of the posterior communicating artery. (*B*) Second right carotid angiogram. There is now only slight residual narrowing of the internal carotid artery and no arterial corrugations are apparent. The intracranial branches now appear normal in caliber. The infundibulum is larger and the posterior communicating artery has filled.

DISCUSSION

Detailed study of the entire series strongly indicated that this phenomenon is an unique entity and a specific arterial response. The remarkable regularity and smoothness of the arterial deformity clearly distinguishes this phenomenon from such conditions as atheroma and fibromuscular hyperplasia, though there is occasionally a very superficial resemblance. Szilagy[10] and others have confused the typical appearance of fibromuscular hyperplasia with the stationary wave phenomenon.

The arterial configuration also appears to be quite distinct from the diffuse spastic arterial narrowing commonly encountered, and from the more localized, more or less severe, circular spastic contractions that may be encountered following severe mechanical irritation of the arterial wall, such as can be produced by catheter manipulation[6] and by intramural or perivascular injection of contrast medium.

Three theories have been advanced to explain this unusual arterial appearance (Table ii). Theander,[11] noting that the waves occurred proximal to some obstructing vascular lesion, but only when collateral circulation was not plentiful, postulated that the longitudinal arterial pulse pressure waves were reflected at a site of obstruction, to produce "standing waves," which

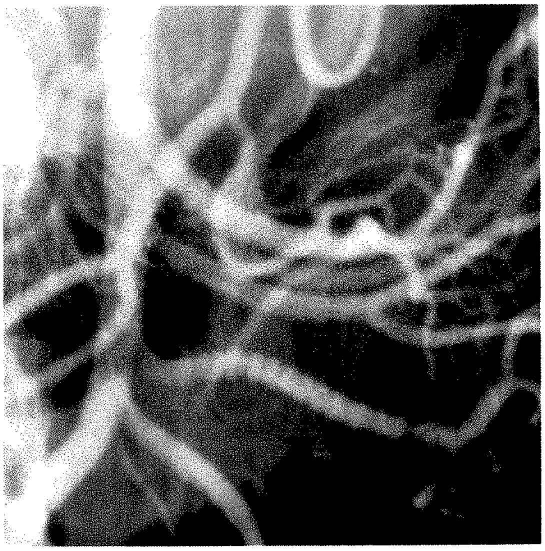

Fig. 5. A 46 year old male with retroperitoneal lymphoma. A superior mesenteric angiogram by selective catheterization showed stationary waves in 4 branches of the superior mesenteric artery, involving segments several centimeters long. At the beginning of angiography 3 branches were strikingly involved and one only slightly. A little later in this serial angiography a fourth branch gradually developed waves and an increase was visible in the prominence of the waves in the other 3 branches during this series. A second injection and serial study showed waves in all of the 4 branches at the beginning and persisting throughout. However, the waves changed slightly in degree during the series. The branch most markedly deformed is indicated by the arrow.

became progressively amplified by resonance, resulting in alternate segments of widening and narrowing of the involved artery.

Wickbom and Bartley[12] included a single example in their group of cases exhibiting arterial spasm, and noted that following priscol injection into the artery, the corrugations diminished. These authors believed the phenomenon to be due to a form of arterial spasm.

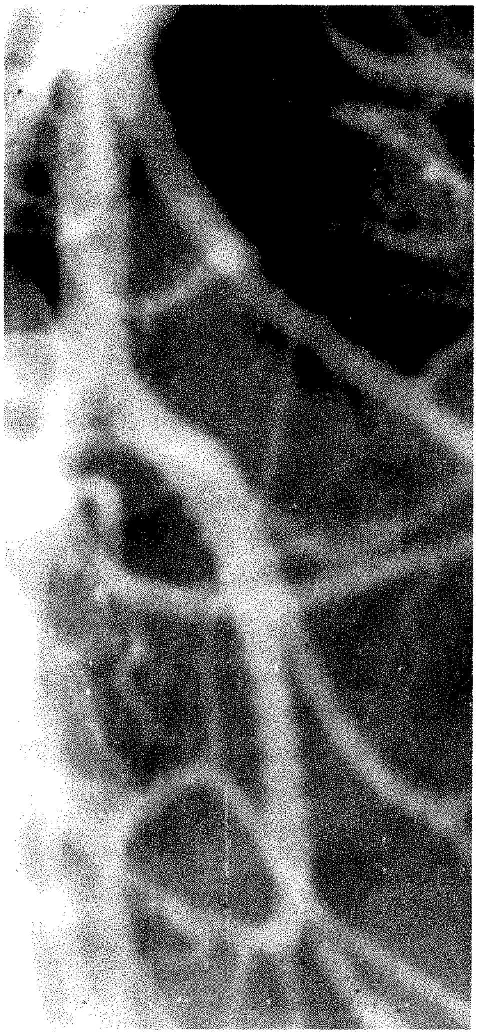

Fig. 6. Injection into the aorta via a transfemoral catheter showed waves in 3 jejunal branches of the superior mesenteric artery over a distance of several centimeters. No definite variation in the wave pattern was evident through the series. Slight wave formation in the superior mesenteric artery between the origins of the involved branches was noted. At the third injection, there was more pronounced wave formation in the same segment of the superior mesenteric artery and now 4 jejunal branches were involved.

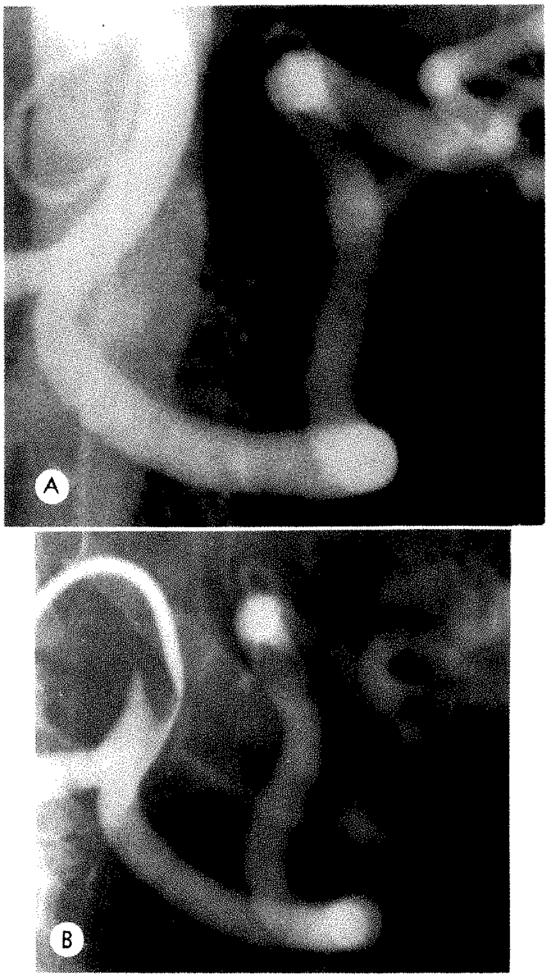

Fig. 7. (*A*) A 74 year old female with metastatic carcinoma of the liver, probably from the colon. A selective celiac catheterization angiogram initially showed no evidence of waves in the splenic artery on 2 injections. (*B*) A further selective celiac angiogram *8 days later* showed very pronounced waves in 2 segments of the splenic artery, separated by a distance of 3 cm. No waves were evident at the inferior loop of the vessel.

Schechter,[6] discussing the incidence of spasm of the internal carotid artery during percutaneous catheterization, included mention of so-called stationary waves. However, his illustrations indicate that his findings represent more or less severe focal

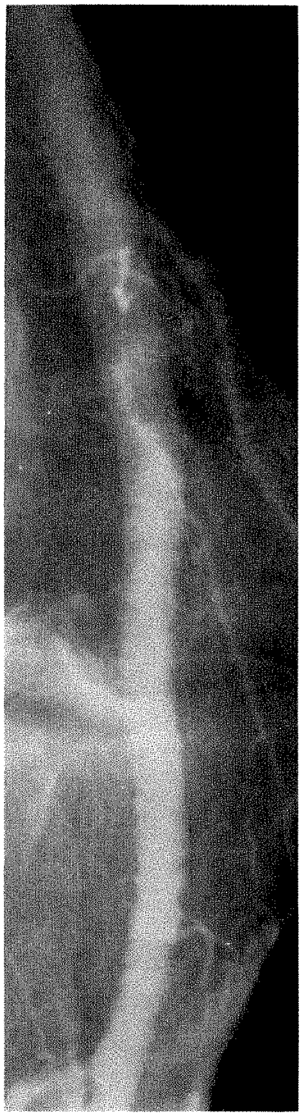

Fig. 8. A 42 year old male with chronic renal failure who was subjected to vascular study before renal transplantation. Arterial waves were demonstrated over an 8 cm. segment on the popliteal artery and there was slight generalized narrowing through the entire segment.

and generalized spasm, rather than the phenomenon under consideration.

Mayall[4] discounted Theander's theory regarding pulse pressure wave reflection at a site of branching or obstruction on the grounds that the pulse pressure wave length is considerably greater than the entire length of the arterial tree and that the energy of reflected waves is probably very small after the second reflection. Present evidence indicates that the pulse pressure wave undergoes only one major reflection at the periphery, where the ratio of reflected and incident waves is of the order of 0.3 to 0.5.[2] Owing to marked dampening in travel, due to viscosity of blood and the structure of the arterial wall, Mayall considered that it was unlikely that the energy of reflected waves was sufficient to produce standing waves in any part of the arterial tree, even with the possibility of resonance. He pointed out that studies have indicated that the primary longitudinal arterial pulse waves produce no roentgenographically measurable change in the diameter of the arterial lumen.[5] He found no measurable change in the diameter of a freshly excised arterial segment when the intraluminal pressure was raised from 100 to 300 mm. of mercury.

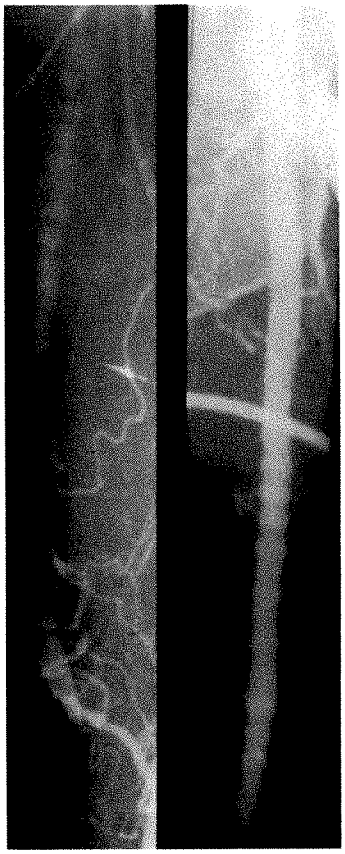

Fig. 9. Distal (left) and proximal (right) femoral artery.

Table I

Authors		No. of Examples	Artery Involved	Material
Wickbom and Bartley[12]	1957	1	femoral	108 extremity angiograms
Theander[11]	1960	4 ?	femoral and profunda femoris also iliac	217 lower extremity angiograms
Sutton[7]	1962	approximately 12 occasional 1 1	femoral iliac internal carotid renal	not known
Schechter[6]	1963	?	internal carotid?	over 1,000 carotid angiograms
Mayall[4]	1964	1	superior mesenteric branches	
Present Series		6	internal carotid	2,000 carotid angiograms
		1 1 2 1	femoral popliteal superior mesenteric and branches splenic*	not known

* Apparently reported for the first time in this artery.

Mayall suggested that the arteriographic appearance of standing waves was artifactual and due to the formation of ripples between layered contrast material and overlying blood when viewed at right angles to the interface. He suggested that rapid acceleration and deceleration of blood flow during the cardiac cycle produced slipping between the 2 layers, due to the different viscosity and density of the fluids, resulting in ripples at the interface. He felt that this would be more likely to happen when the late diastolic backflow in an artery was increased by arterial obstruction and that vasodilatation resulting from priscol injection resulted in a reduction of the backflow phase in the artery, which would tend to cause the ripples to disappear.

Of the theories presented, that of Mayall can most readily be discounted. One cannot doubt that the phenomenon, as seen in different portions of the arterial tree, is essentially of the same nature and the carotid angiograms in the present series (and the one illustrated by Sutton[7]) clearly indicate that layering has not occurred (the lateral studies having been made using a horizontal beam) and is, therefore, not an essential prerequisite to the appearance of the wave phenomenon. In addition, the

Table II

THEORIES

Theander: Longitudinal arterial pulse pressure waves reflected at point of obstruction and resonance-amplified. (Frequency of associated obstructive arterial disease; lack of occurrence with good collateral circulation)

Wickbom and Bartley; Schechter: Circular spastic muscular contractions

Mayall: *En face* view of ripples at horizontal interface between layers of contrast medium and blood, caused by different rate of change of velocity when arterial flow rate suddenly increased or decreased; due to different viscosity and density of blood and contrast material illustration by Mayall of his experimental results indicates that the contrast medium irregularities produced are quite different from the configuration of "stationary waves."

On the other hand, there is considerable *circumstantial* evidence to suggest that these arterial corrugations are a result of a type of unusual circular muscular contraction of a spastic nature in the arterial wall. In many instances, it is possible to detect a *generalized* narrowing of the arterial wall through the involved segment. As in the case of common varieties of spasm, the condition is most frequently seen in younger adult patients. In general, the involved arterial segments appear otherwise healthy. The condition occurs in peripheral arteries having a well-developed muscle layer which are known to be prone to spastic contraction when irritated by various means. Generalized spastic narrowing and corrugations not infrequently coexist in the same or in adjacent branches and may appear sequentially in the same artery. Occasionally, there is a visible increase in the extent of the waves in one or more arterial branches with further contrast injections during an angiographic procedure.

In the internal carotid artery, the condition apparently tends to be associated with subarachnoid hemorrhage (which had occurred in each of the 3 cases reported here), in which condition there is increased arterial irritability and a tendency to spasm. However, the finding of stationary waves 9 months following the hemorrhage in Case II is not readily explained on this basis. In the carotid system, waves appear not to involve the carotid sinus or the common carotid artery, both of which are usually spared in spasm of this portion of the arterial tree. On the other hand, the phenomenon has not been seen in the intracranial portion of the internal carotid artery, a segment frequently the site of spasm.

The condition has not been described in the vertebral artery, a vessel in which spasm is rarely, if ever, visualized.

Against the Theory of Spasm. The remarkable regularity or "periodicity" of the

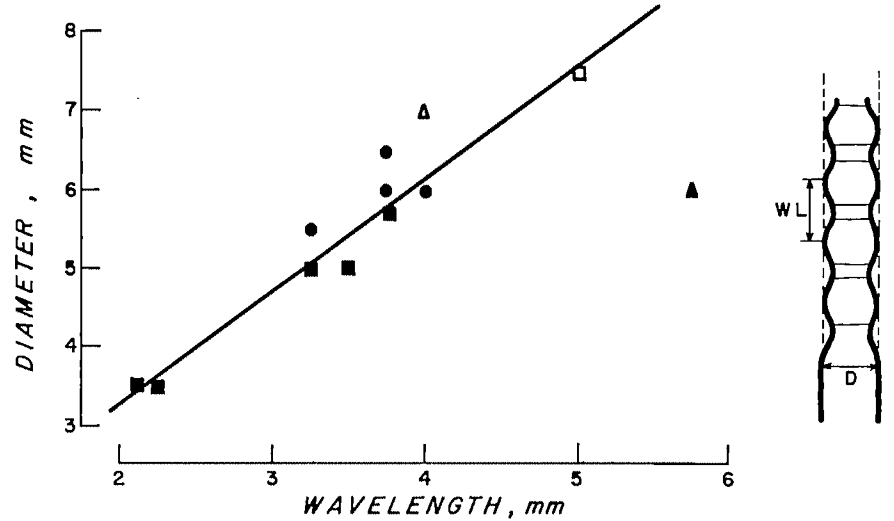

FIG. 10. The wavelengths of the corrugations in the involved arteries have been plotted against the diameters of the respective arteries taken just proximal to the segments involved.

● internal carotid artery (Cases I and II. Reliable measurements could not be obtained in Case III.)
△ popliteal artery
■ superior mesenteric branches
□ splenic artery
▲ femoral artery (64 year old male with severe generalized arteriosclerosis; see text.)

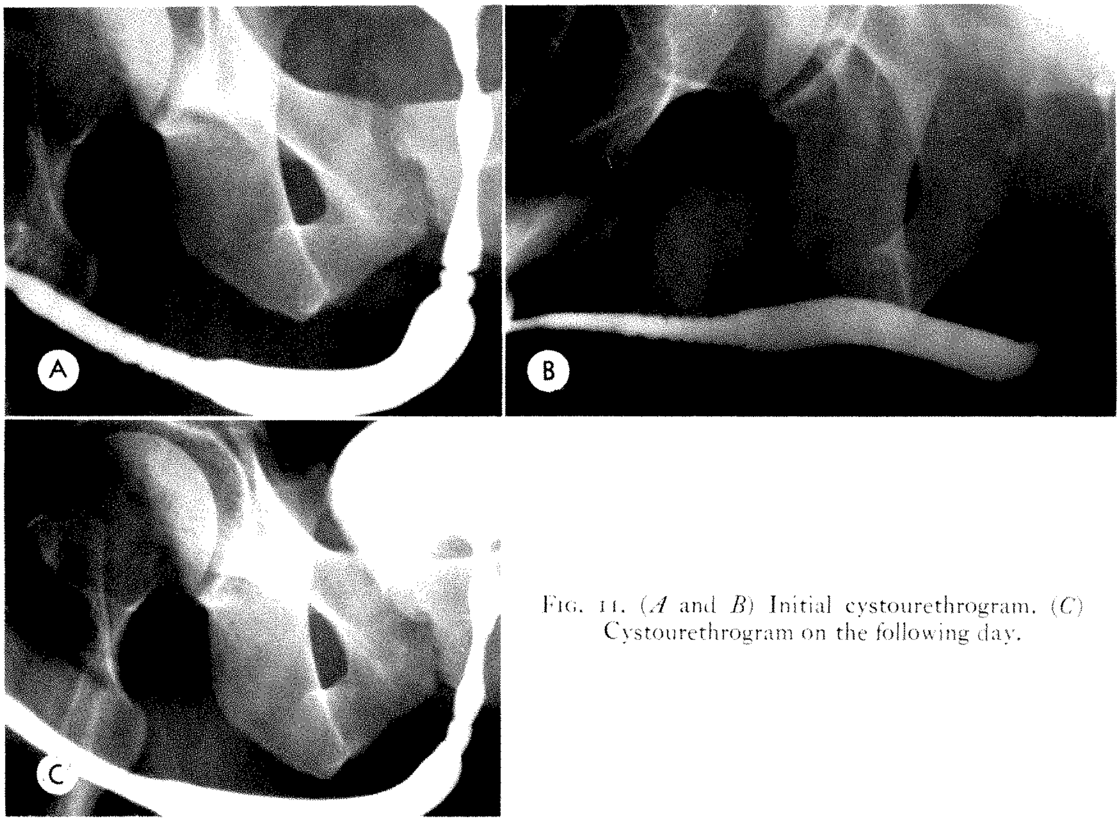

Fig. 11. (*A* and *B*) Initial cystourethrogram. (*C*) Cystourethrogram on the following day.

undulations of the vessel lumen, with a high degree of symmetry around the entire circumference of the artery involved, is in striking contrast to the irregular contractions of the artery wall seen in well documented examples of segmental arterial spasm. This marked regularity is seen over long arterial segments and contrasts with the irregular muscular contractions occasionally encountered in the arterial tree and in other muscular tubular organs. These features suggest a fundamental difference between the wave phenomenon and muscular spasm.

The regularity of the phenomenon, on the other hand, is highly compatible with a passive mechanical deformation, such as might be caused by resonance of arterial pressure waves, reflected from a point of arterial obstruction or a site of high peripheral resistance, as in the case of considerably elevated intracranial pressure commonly present immediately following acute subarachnoid hemorrhage. It is interesting to note that these waves have not been reported in the larger intracranial arteries, in which diffuse spasm is frequently seen (as in Case III of the presently reported series). This may be due to the thinner walls of intracranial arteries, but also suggests that the waves are not due to spastic muscular contractions.

Measurement of the wave length of the stationary waves in arteries of various calibers in the collected cases shows a good correlation with arterial diameters (Fig. 10) in all but 1 case (that of femoral arterial involvement in the only elderly patient in the group with advanced generalized arteriosclerotic disease). The reason for this single discrepancy is not clear. Marked loss of elasticity of the arterial wall would be expected to reduce the likelihood of stationary wave occurrence, whether the phenomenon be due to resonance amplification of reflected arterial pressure waves or to spastic contractions of the artery, and, if due to the former, alteration of the frequency of the waves would not be anticipated.

No correlation was found between the amplitude of the waves and arterial diameters, or between wave length and amplitude. If amplification by resonance of the reflected pressure waves is required before the arterial wall is thrown into undulation, a very particular set of physical conditions would have to be met for the phenomenon to occur. This would explain the low incidence of the finding. The action of vasodilators, in decreasing peripheral resistance, could explain those cases in which arterial waves were abolished or reduced by this means.

In none of the cases reported here was there an opportunity to examine the involved arterial segments by surgical exposure. Previous reports of the phenomenon have not mentioned direct examination of the arterial walls in the affected segments.

One of the patients included in this series succumbed to lymphoma some time following abdominal angiography. The mesenteric arteries were unfortunately not studied by postmortem angiography, but serial sections of the mesenteric arteries revealed no pathologic changes.[1]

During the course of the present study, a voiding cystourethrogram was discovered which demonstrated a wave pattern in a segment of the cavernous portion of the urethra, a highly elastic organ *devoid of muscle* (Fig. 11, *A* and *B*). The wave pattern was seen in cystourethrograms obtained both during and after voiding against meatal occlusion. The wave pattern presented a remarkable resemblance to that seen in arteries. Another cystourethrogram made on the following day showed no waves during voiding against a closed meatus (Fig. 11 *C*). Thus, the functional nature of the phenomenon was clearly indicated. Obviously, the appearance in this organ cannot be ascribed to muscular action. On the other hand, sudden voiding against a closed urethral meatus would be expected to produce conditions under which a pronounced reflection of a pressure wave could occur. In a very elastic tubular organ, such a pressure wave could well undergo resonance to produce the effect noted. The ratio of the wave length to diameter corresponds closely with that found in the arterial corrugations studied.

SUMMARY

Six angiograms (of 3 patients) showing stationary wave formation involving the internal carotid artery were found on detailed review of 2,000 carotid angiograms, an incidence of 0.3 per cent. In general, the carotid changes were less striking than those seen in many other peripheral arteries involved in other patients. Subarachnoid hemorrhage had occurred in each patient. These patients were all young adults, each in the third decade.

A series of roentgenograms of this condition in other arteries, including an example in the splenic artery, involvement of which has not previously been recorded, confirms the striking similarity of the arterial configuration, independent of the artery involved. The findings indicate that the phenomenon is not an expression of segmental arterial spasm and evidence is presented to confirm the theory of Theander, that the appearance is due to a passive change in the arterial wall resulting from reflection of the arterial pressure wave, amplified by resonance.

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A NEW INSTRUMENT: CONTROLLABLE GUIDE FOR ANGIOGRAPHY*

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CONTINUING progress in technical developments relative to selective vascular examinations has occurred since Seldinger[1] first described his method for percutaneous arteriography. The incorporation of certain opacifying agents within the walls of low density polyethylene and polytetra fluorethylene (teflon) catheters simplified the fluoroscopic approach to most major vessels; however, a small group of vessels, including the coronaries, bronchials, inferior mesenteric and adrenals, requires more catheter control for uniformly successful examinations. With this in mind, we have constructed a self controlled guide wire adaptable to all catheters (Fig. 1, *A* and *B*).*

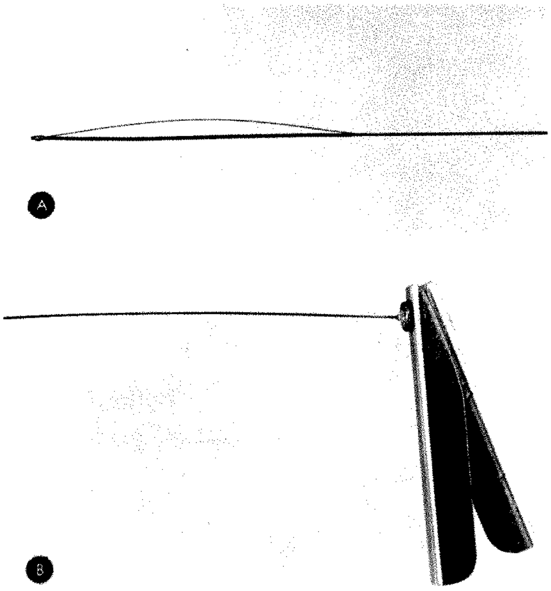

FIG. 1. Controllable guide instrument. (*A*) Tip of guide demonstrating tubing and inner wire. (*B*) Handle of the guide is detachable.

The basic instrument allows the catheter tip curve radius to vary from 0.5 inch to approximately 2 inches (Fig. 2). In addition catheter rotational recoil is eliminated since the guiding mechanism allows a 1.1 ratio between the turning of the external catheter guiding mechanism and the turning of the internal direction of the catheter tip. The main body of the catheter guide is a .018 inch or .025 inch in diameter 304 stainless steel tube of either 50, 80, or 100 cm. length, depending on the catheter used. The inside diameter of this tubing is .010 inch allowing for the insertion of a .007 inch stainless steel wire which is used to apply force to the tip flexing member, causing this member to bend in the direction of the free wire.

One end of the .007 inch wire is extended approximately 2 inches beyond the catheter body tubing and silver-soldered to a second piece of stainless steel tubing which is laid back along the force wire and silver-soldered to the catheter guide body. The lower end of the catheter guide body is fitted with a small sleeve which is silver-soldered to the catheter body and the entire assembly is screwed into a matching hole on the face of the upper level plate (Fig. 3).

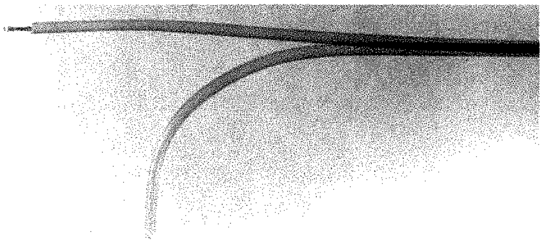

FIG. 2. Double exposure showing tip of controllable guide at tip of catheter in neutral position. Pressure applied to proximal lever results in controlled flexion. When guide is withdrawn from the catheter tip 1 to 2 cm., the distal limb of catheter curvature is increased.

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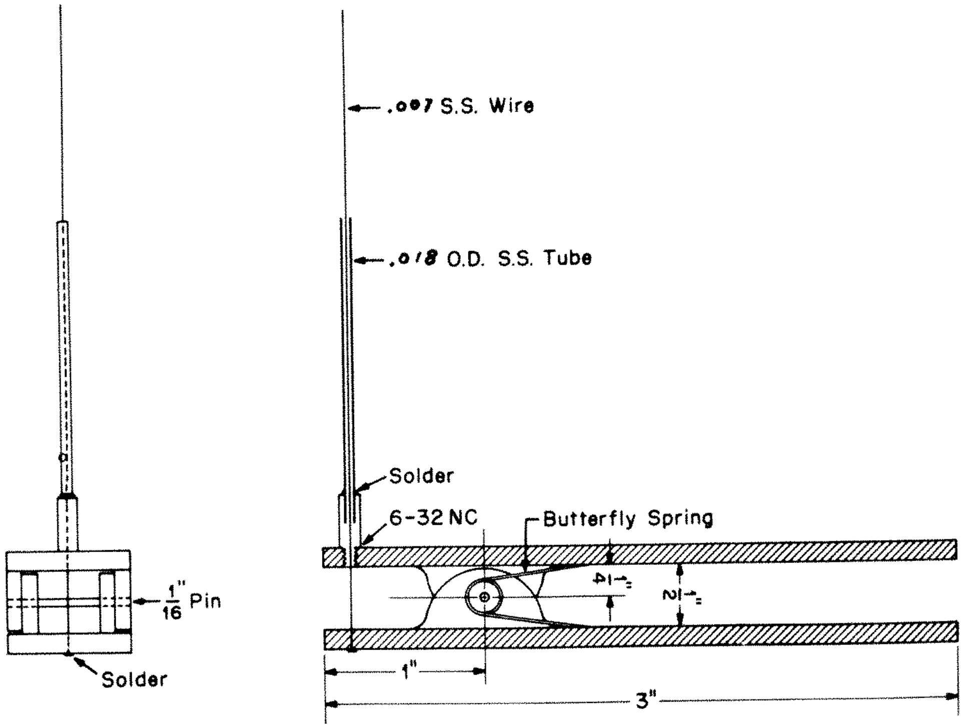

Fig. 3. Line drawing of controllable guide instrument.

The force wire extends through the sleeve and the upper lever plate to a hole bored in a matching lower lever plate where it is mechanically fastened to the lower plate and proper tension is fixed. When bending of the catheter tip is required, the lever plates are compressed. This pulls the force wire, causing the flex arm to bend. If the bend is not in the proper direction, the lever plates are released. When the assembly is turned in the proper direction, the lever plates are compressed again. When the catheter is in place, a small amount of contrast medium is injected for position accuracy and the guide wire may then be removed. In the event that the guide wire is damaged, it may be released from the actuating lever and another inserted. Experience has shown that, in order to maintain flexibility and yet retain spring action, tubing of .018 to .025 inch diameter is the most satisfactory of the materials evaluated.

Figures 4, 5 and 6 demonstrate the application of the guide instrument in a red Ödman catheter. In addition to being able to control the catheter, for insertion into inaccessible vessels it is also feasible to utilize smaller thin walled catheters in the range of 2 mm. external diameter. Experience has shown that complications at the puncture site occur less often with the smaller catheter systems and, consequently, the ischemic effects of larger catheters

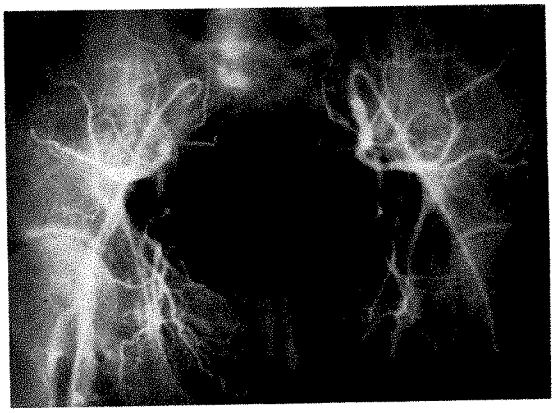

Fig. 4. Selective bilateral internal iliac arteriogram with catheters in position. Early arterial phase in a case of carcinoma of the bladder floor.

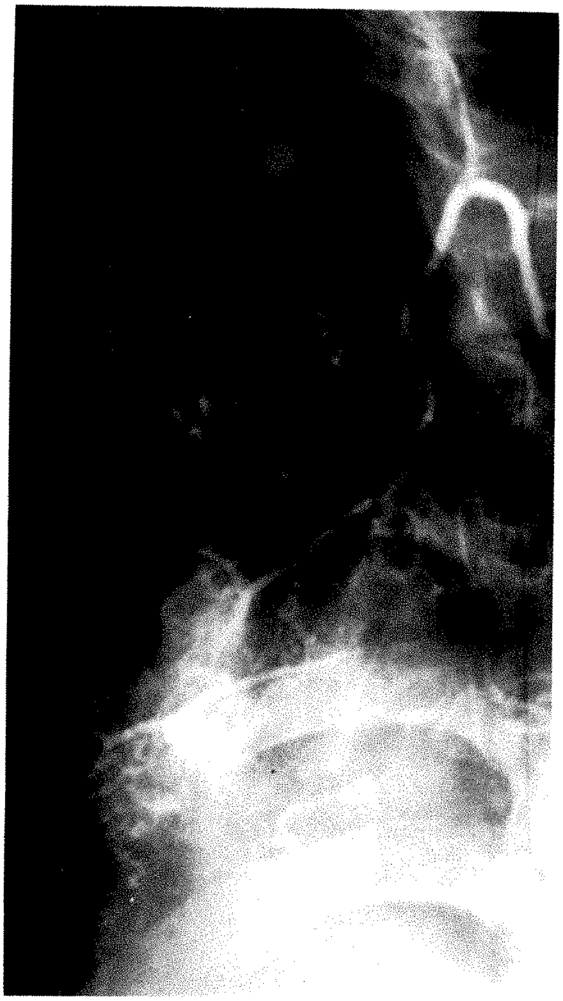

FIG. 5. Selective bronchial arteriography in a case of proven bronchogenic carcinoma. Note tumor circulation in the mass adjacent to the right hilus.

within branch vessels can be avoided. This has particular application in the coronary, bronchial and inferior mesenteric circulation. With the bronchial and inferior mesenteric circulation, the red Ödman catheter is inserted via the femoral artery. For the coronary circulation, the axillary artery is punctured and a red Ödman or small 160 polyethylene catheter may be used. Because of the soft flexible tip of the polyethylene catheter, there is essentially no trauma to the coronary ostia.

Recently we have modified the control handle, as illustrated in Figure 7.

Additional applications of the guide wire include vertebral artery catheterization, control of the catheter so that the descending aorta may be entered when being approached from a tortuous arch after axillary insertion, the left common carotid artery from the aortic arch, selective external and internal carotid examinations, subsegmental hepatic arteriography as well

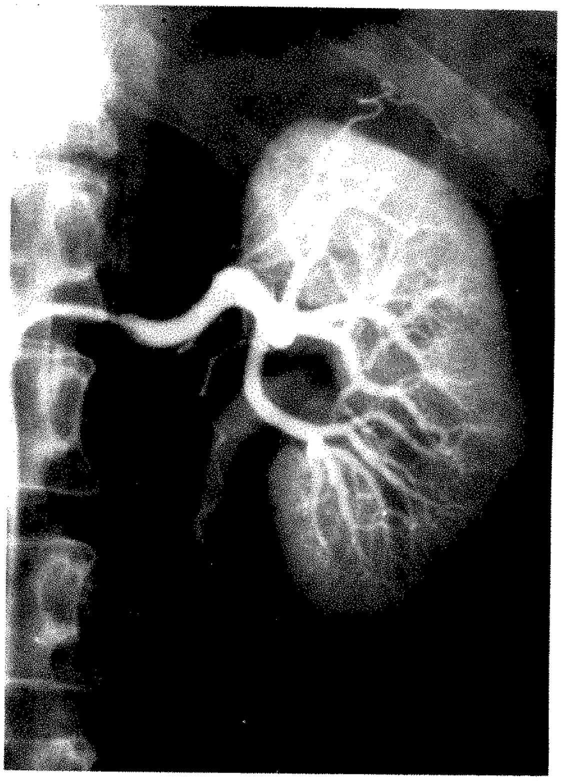

FIG. 6. Normal selective renal arteriogram.

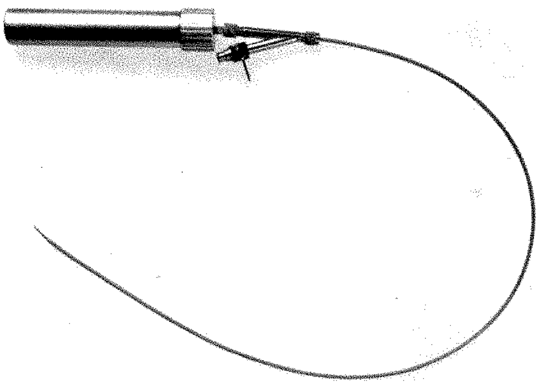

FIG. 7. Modified control handle and guide assembled in a red Ödman Ledin catheter.

as adrenal venography by catheterization of the adrenal vein.

SUMMARY

A new controllable guide adjustable for use in all intravascular catheters has been constructed. The purpose has been to simplify the approach in most catheterization procedures, allowing complete catheter control and ultimately decreasing the necessity for large thick walled catheters for the selective vessel examinations.

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TIMING DEVICE FOR CINEANGIOCARDIOGRAPHY*

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SELECTIVE angiocardiography has become an integral part of cardiac catheterization during the past decade. This procedure has been used primarily for (1) precise clarification of the anatomic nature of cardiac abnormalities and (2) gross detection of abnormal circulatory pathways. Recent studies by Dodge and co-workers,[1] Wood *et al.*,[3] and studies in progress in our laboratory[2] have scored the value of analysis of angiocardiographic data for studying cardiac hemodynamics.

Cine-studies of the timing of intracardiac shunts and analysis for changes in left heart volumes require precise timing of the individual frames as related to the cardiac cycle. In this report a simple timing device for accurate recording of individual cine-frames for such studies is described. Biplane cineangiocardiography is used for contrast studies in our laboratory, but it must be emphasized that the timing device is equally practical for any uniplane roentgen-ray system. Additional recording equipment includes a multichannel photographic and magnetic tape recorder. During the filming of biplane cineangiocardiograms, the electrocardiogram, pressure tracings and roentgen-ray pulses of the anteroposterior and lateral cine-frames at 60 per second are recorded simultaneously.

A schematic drawing of the phototube and accompanying circuitry is presented in Figure 1. The RCA 922 vacuum phototube is covered with a zinc sulfide fluorescent screen and the entire device is enveloped with insulating tape. It is positioned in the fringe of the roentgen-ray beam, preferably between the focal spot and shutter such that it does not interfere with cine-filming. With each roentgen-ray pulse, the phototube output of 0.2 millivolts is fed to the output of an A-C coupled differential amplifier. Due to the distance between the roentgen ray and recording equipment, a lengthy (20 foot) cable is required to connect the phototube device with the recording unit. If biplane studies are used, a separate phototube unit is required for the vertical and horizontal axes.

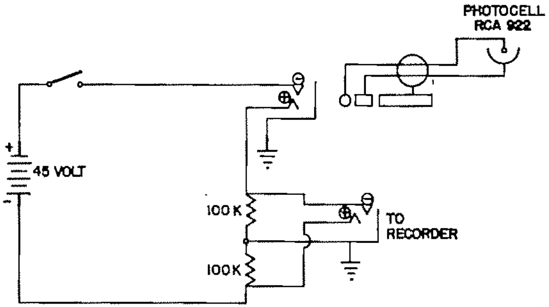

FIG. 1. Circuitry for phototube device. An RCA 922 phototube is powered by a 45 volt battery. The 0.2 millivolt output of the device is recorded via push-pull connections to an A-C coupled pre-amplifier (*e.g.*, a rapid response electrocardiograph). Two such units are utilized for the biplane cineangiocardiography and the entire circuitry can be enclosed in a unit as small as 3×3 inches.

The timing of the data recorded with these units is illustrated in Figure 2*A*. During cine-filming, the electrocardiogram, right ventricular pressure, and pulses corresponding to the individual cine-frames of the two units are recorded photographically at a slow paper speed (10 mm. per second) and on magnetic tape. Following the study, the data are played back for time expansion. Figure 2*B* shows the facility with which the individual cine-pulses can be correlated with the exact

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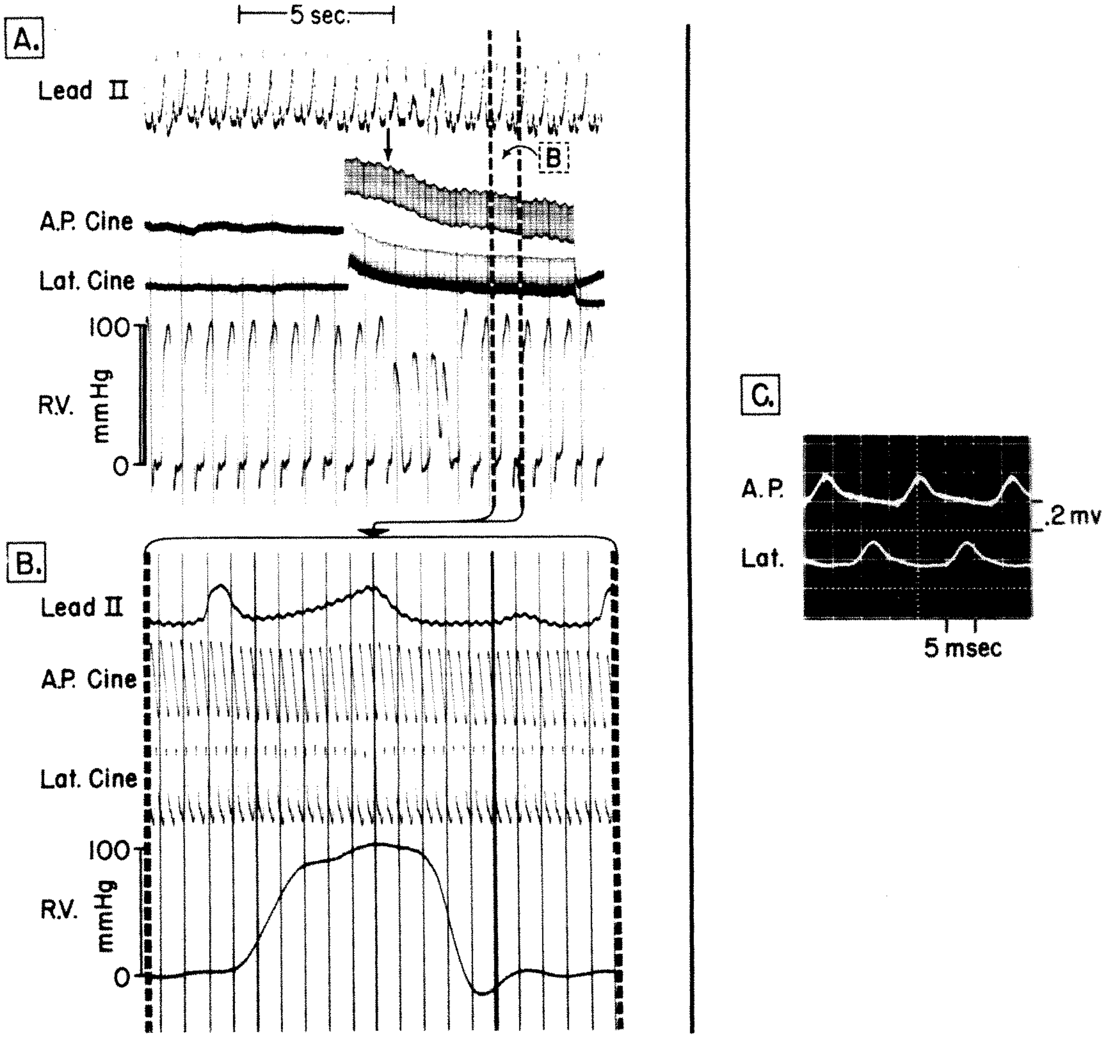

Fig. 2. Data recorded during biplane cineangiocardiography. Catheters were positioned in both right and left ventricles for selective left ventricular angiocardiography. (*A*) The outputs of the anteroposterior and lateral phototube devices were recorded simultaneously with the electrocardiogram and right ventricular pressure. The arrow indicates the onset of the injection of contrast medium. Premature ventricular beats occurred coincident with the injection. (*B*) The data recorded in *A* were stored on tape and played back for time expansion (200 mm./second). The cardiac cycle shown illustrates the facility with which individual frames can be related to cardiac events. (*C*) Oscilloscopic tracing of phototube outputs with alternate firing of the anteroposterior and lateral cine-units at 60 pulses per second.

time of the cardiac cycle during which each frame was exposed. There was no significant pulse-time delay (less than 1 microsecond) through the lengthy cable, electronic system and image intensifier tube. This was verified by comparison with another phototube system located at the output phosphor and connected directly to an oscilloscope, thereby bypassing the lengthy cable and electronics.

Since the cine-magazine reaches full speed prior to initiation of roentgen-ray pulses, the number of recorded pulses and corresponding number of cine-frames are always equal in number. Figure 2*C* is an oscilloscopic tracing of the wave form output from both phototubes during biplane cineangiocardiography with alternate firing of the anteroposterior and lateral cine-units. Note that the recorded pulses

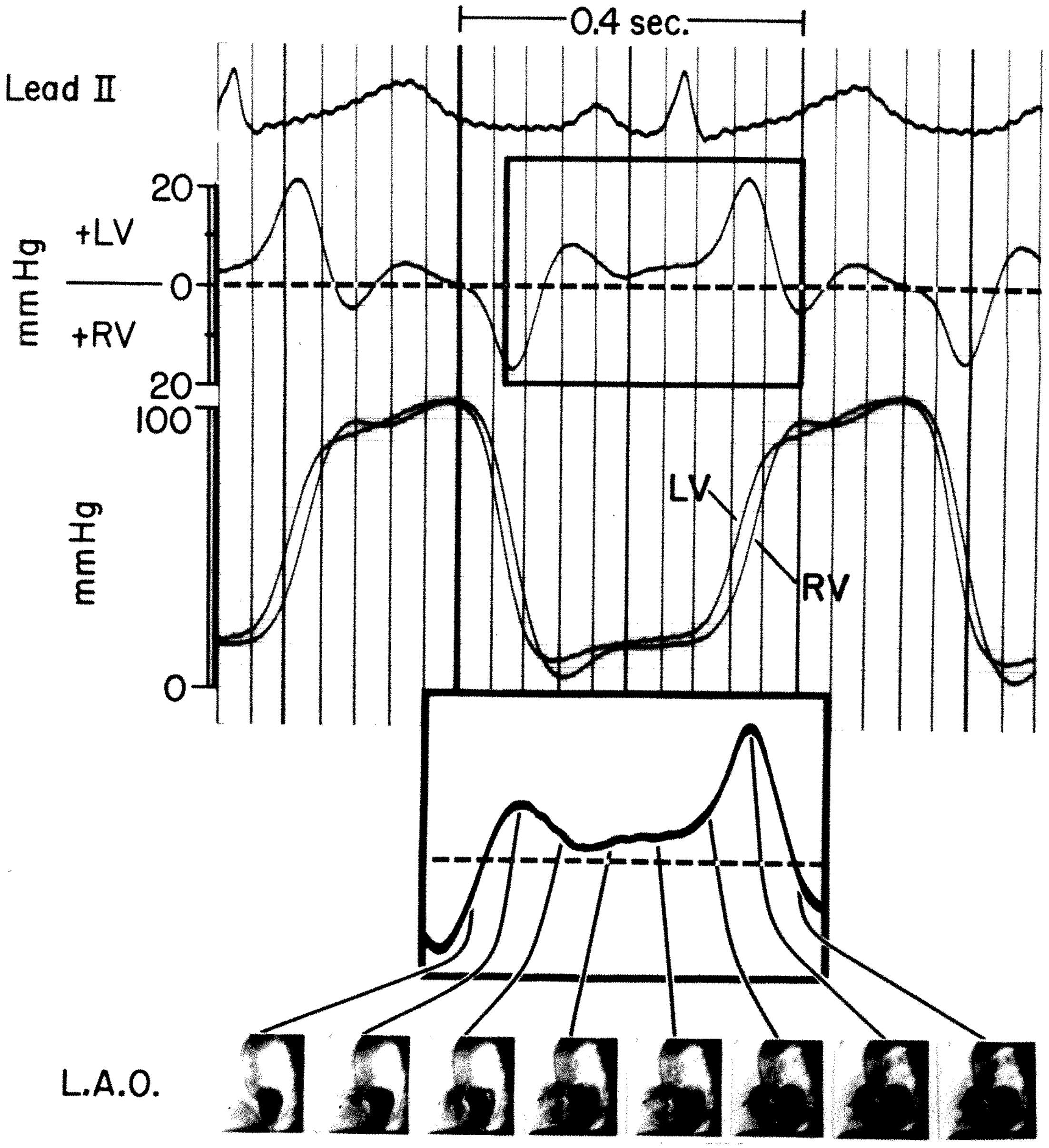

Fig. 3. Correlation of cine-frames to the cardiac cycle. Lead II of the electrocardiogram is shown at the top. The bottom two lines represent simultaneous right and left ventricular pressures. The middle graph is the resultant pressure gradient between the right and left ventricles. The boxed-in part of the gradient is shown below. Each cine-frame is correlated to the pressure gradient as it occurs during the cardiac cycle by means of the phototube timing device.

from the two units are 90° out of phase.

Figure 3 illustrates the practical application of the phototube timing device. Eight cine-frames are shown of a child with tetralogy of Fallot with injection in the left ventricle, left anterior oblique position. Beginning on the second cine-frame, the initial left-to-right shunt through a ventricular septal defect was noted and a second left-to-right shunt was noted beginning on frame No. 6. By correlating each cine-frame with its corresponding pulse (indicated in Figure 2), one can relate any cine-frame to its specific part of the cardiac cycle. The boxed-in area represents the pressure gradient of the left and right ventricles during isometric ventricular relaxation, ventricular diastole and isometric ventricular contraction. Therefore, the left-to-right shunts occur during early diastole and isometric ventricular contraction.

SUMMARY

A simple phototube timing device is described to relate individual cine-frames to the particular part of the cardiac cycle during which they occur. A practical application of the use of this device is also illustrated.

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A CURVED-TIP GUIDE WIRE FOR THORACIC AND ABDOMINAL ANGIOGRAPHY*

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TORTUOSITY and atherosclerosis, especially of the iliac arteries, frequently prohibit the retrograde passage of a standard guide wire or catheter in elderly or hypertensive patients. Recently, Baum and Abrams[2] described a catheter whose tip was formed to conform to the letter "J". Because its leading edge was smooth, it readily negotiated tortuous arteries otherwise impossible to traverse.

The major disadvantage of the J-shaped catheter is the need to replace the angiographic catheter with the J-catheter if an obstruction is encountered. Once the obstruction is traversed, a second change is necessary, changing back to the angiographic catheter from the J-catheter. With each catheter change, there is some chance of additional arterial trauma or, particularly, of hematoma formation.

In this communication a curved-tip guide wire which has all the advantages of a "J" tip catheter, but has the important additional advantage that a catheter change is not required each time an obstruction is encountered is described.

THE CURVED-TIP GUIDE WIRE

The curved-tip guide wire* consists of a standard coiled spring guide wire, currently used with the Seldinger technique,[3] in which the terminal 1.5 cm. of the 6 cm. flexible segment assumes a curved configuration 8 mm. in diameter (Fig. 1). The curved flexible portion of the wire easily straightens while passing through the needle or catheter, but once within the vessel it reassumes a J-shape. Its leading edge is thus converted into a smooth flexible segment with all the uses of the J-

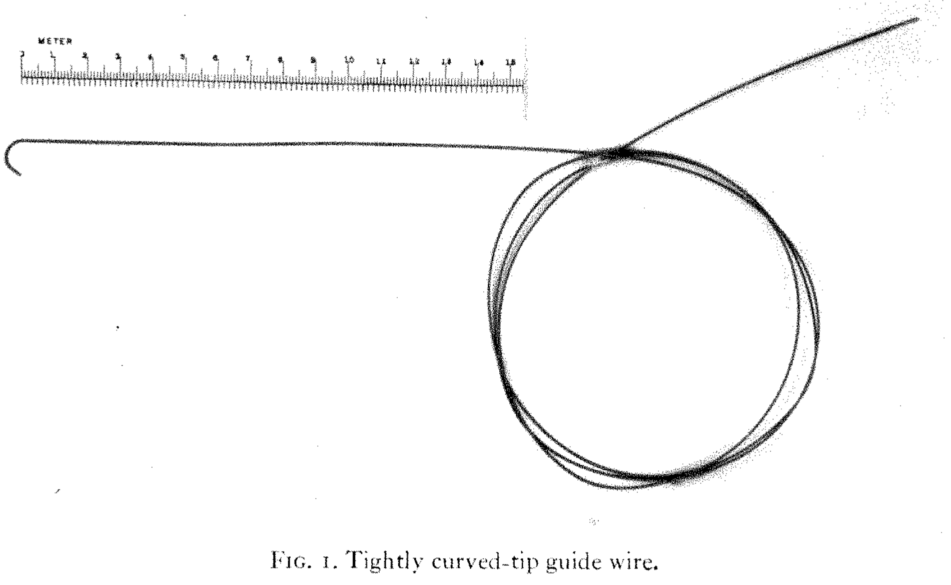

FIG. 1. Tightly curved-tip guide wire.

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shaped catheter, but with the additional advantage that catheter changes are not necessary. The technique employed with this wire is simply to pull the tip of the catheter back a short distance from the obstruction, pass the guide wire through the catheter until the flexible curved tip of the guide wire has passed completely through the tip of the catheter and reassumed its J-curve, pass the curved guide wire and the catheter along with it by the obstruction, and then remove the curved-tip guide wire.

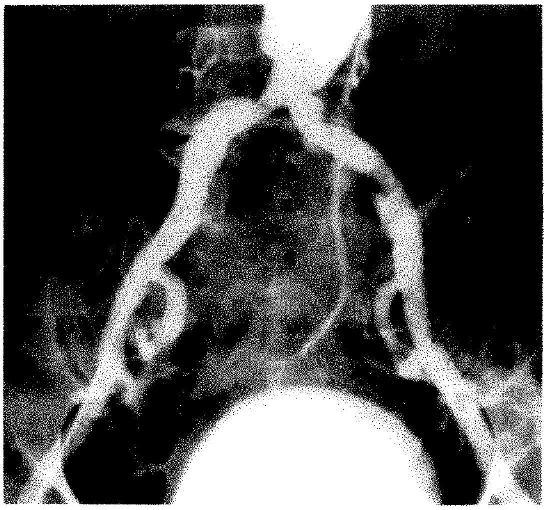

Fig. 2. Angiogram of iliac artery in which obstruction was encountered with catheter but which was easily traversed by curved guide wire.

The curved-tip wire has been useful in traversing tortuous and atheromatous iliac arteries during retrograde catheterization of the abdominal aorta (Fig. 2), and in negotiating passage into the thoracic aorta via axillary artery puncture. This curved-tip guide wire has also proved valuable in severe atheromatous disease where a straight wire or catheter constantly engages atheromatous plaques. It has also been useful when severe atheromata in the aorta prevent further advancement of a catheter during selective catheterization of the branches of the abdominal aorta. During attempts at selective catheterization, searching for the orifice of the desired branch vessel may require repeated ascending and descending passes of the catheter tip by the area of origin of the vessel. In the atheromatous aorta, the catheter tip may be arrested in an upward passage, and

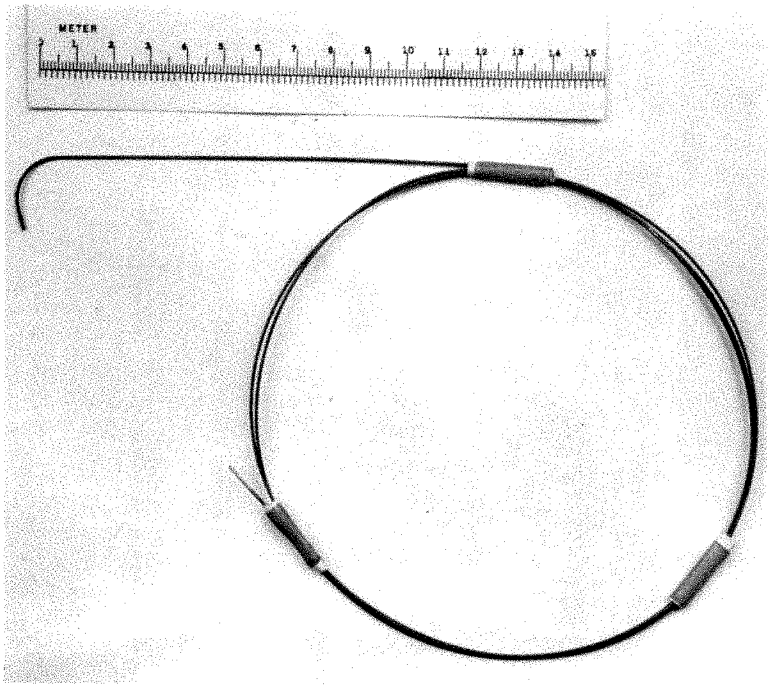

Fig. 3. Widely curved-tip guide wire.

even the introduction of a straight guide wire may not be successful in further advancement. In these cases the utilization of a curved-tip guide wire has allowed advancement upward to the desired position above the orifice of the branch vessel to be catheterized. We formerly employed a "J" shaped catheter in this situation, but found that although only a modicum of additional time was needed for a single catheter change, the multiple changes which were, on occasion, necessitated by difficult selective branch vessel catheterization of a severely atherosclerotic aorta consumed considerable time.

In elderly patients a catheter introduced from the left axillary artery frequently passes into the ascending aorta rather than the descending aorta as desired. A guide wire with a widely curved tip (Fig. 3), similar to that first described by Amplatz and Harner,[1] may enable passage of the catheter down the descending thoracic aorta. We have not, however, found this particular type of guide wire useful in negotiating a tortuous atheromatous subclavian or iliac artery, as the leading point is usually the sharp tip of the wire.

Curved-tip guide wires, with either tight or wide curved tips, have also frequently assisted in guiding the tip of the catheter from the selectively catheterized celiac axis further on into the splenic, hepatic and left gastric arteries. Even selective catheterization of more distal branches of these vessels, such as the gastroduodenal artery, has sometimes been aided by the use of curved tip wires.

The tightly curved-tip guide wire has been utilized in over 40 instances of difficult catheter passage and it has been instrumental in successfully obtaining angiograms in the majority. Gentle manipulation and avoidance of forceful movement against resistance have always been the cardinal precepts to avoid the risk of intramural passage with dissection of the arterial wall, of dislodgment of a plaque, or of arterial spasm. By following these precepts, there have been no complications using the tightly curved-tip guide wire.

SUMMARY

A curved-tip guide wire, which is a Seldinger type of guide wire in which the terminal portion of the flexible end is formed into a tight curve, is described. This wire has proved useful during angiographic procedures in negotiating atheromatous and tortuous iliac and subclavian arteries as well as in the aorta when the usual wires and catheters have encountered obstruction. A similar wire with a wider curved tip has aided in passage of a catheter into the descending thoracic aorta from the axillary or brachial approach in elderly patients. Both tight and wide curved-tip guide wires have aided in selective catheterization of the branches of the celiac axis. The tightly curved-tip guide wire can be utilized under the same conditions as a J-shaped catheter but has the great advantage that a catheter change is not required each time an obstruction is encountered.

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THE FLOPPY WIRE AS AN AID IN ARTERIAL CATHETERIZATION*

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IN THE past few years advances in surgical and roentgenologic techniques for study of the vascular system have enabled the surgeon to extend the term "operable case" to a large number of patients of advanced age with extensive vascular disease. The success of surgery depends upon two factors: (1) complete clinical and roentgenographic evaluation of the case, and (2) surgical technique. Roentgenographic evaluation comprises the demonstration of the site and number of lesions, the extent of the block, and the status of the run-off. All this information must be obtained regardless of the age of the patient and the extent of the lesion, if any surgical reconstruction is to be considered.

For this reason we would like to stress the importance of roentgenographic techniques in obtaining such data. The modern radiologist should be well versed not only in the interpretation of the vascular studies but also in the means by which these studies may be obtained.

One of the great difficulties in arterial catheterization in elderly persons is offered by the marked tortuosity of the arteries (iliac, subclavian and aorta) and extensive arteriosclerotic plaques. The use of a straight stiff catheter or a wire with a 3 cm. flexible tip may be dangerous in that a forceful attempt to overcome the difficulty encountered in advancing the wire may result in undesirable situations.

These consist of perforation of the arterial wall already damaged by the disease (Fig. 1A); subintimal insertion of wire and formation of a new channel under an atheromatous plaque (Fig. 1, B and C); dislodgment of an atheromatous plaque with formation of a free embolus (Fig. 1D); and bending of the wire at the junction between the flexible part and the inner core with the possibility of fracture and loss of its flexible tip (Fig. 1E).

To overcome this difficulty without damage to the wall of the artery, many techniques have been suggested. Baum and Abrams[3] suggested the use of a J-shaped polyethylene catheter for a tortuous vessel. Viamonte and Stevens[8] described a special curved catheter with a soft flexible, tapered radiopaque tip and a semirigid body. The curve of the catheter can be varied in degree by advancement and withdrawal of a metallic stylet. The complete advancement of the stylet results in straightening of the catheter and, upon withdrawal of the stylet, the catheter will resume its former shape. Amplatz *et al.*,[1] in describing selective catheterization of the brachiocephalic vessels, mentioned the use of a guide wire with a curved tip and with the flexible part 9 inches in length. Riley, Hanafee, and Weidner[5] reported the use of a double catheter assembly in traversing a tortuous artery and in the catheterization of the

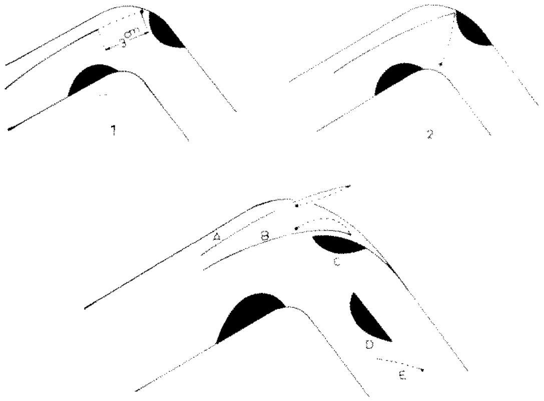

FIG. 1. Sketch showing the bend of the conventional wire when an obstacle is encountered (1, 2) and the complications which may arise if undue force is applied to the wire (A, B, C, D, E—see text).

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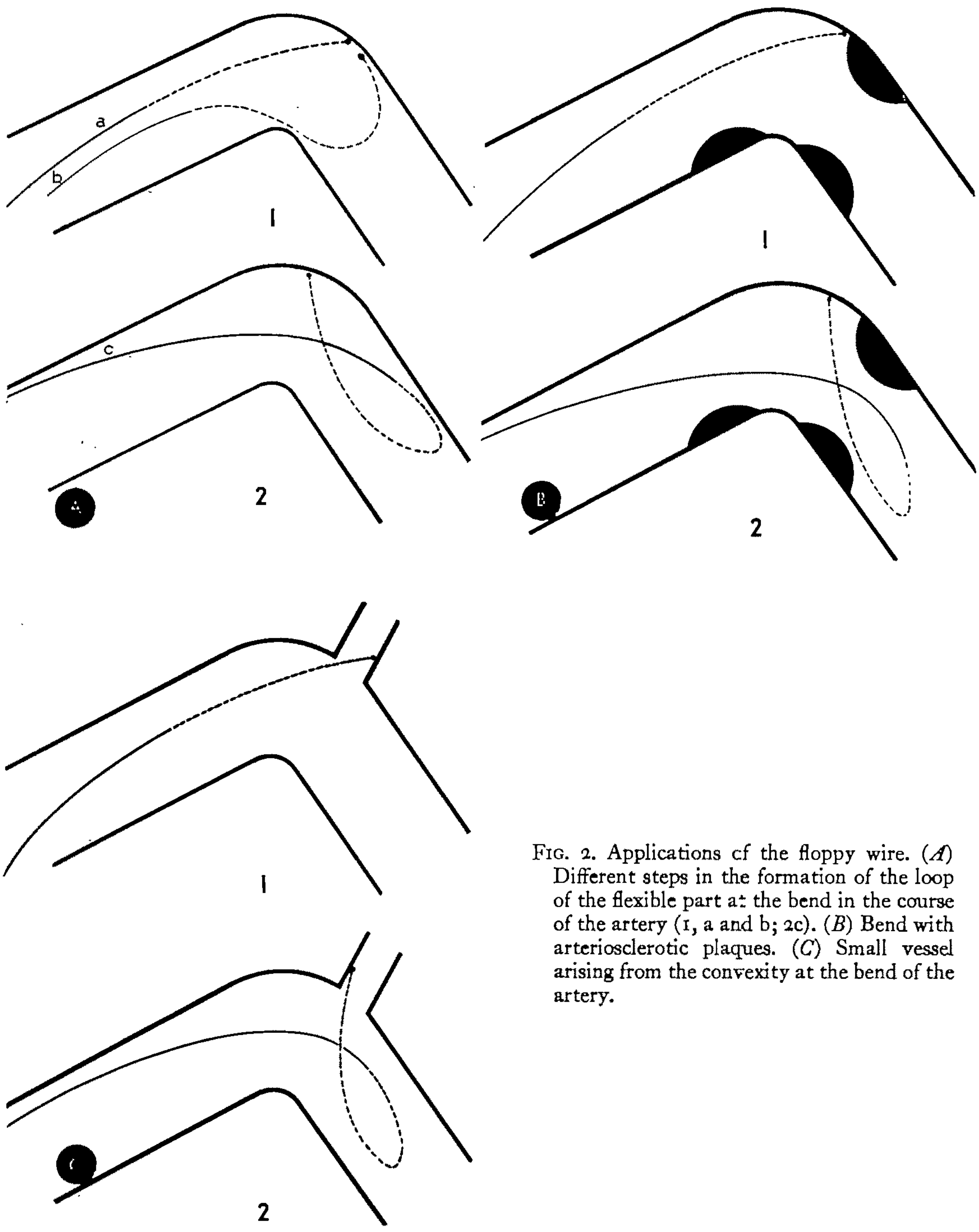

Fig. 2. Applications of the floppy wire. (*A*) Different steps in the formation of the loop of the flexible part at the bend in the course of the artery (1, a and b; 2c). (*B*) Bend with arteriosclerotic plaques. (*C*) Small vessel arising from the convexity at the bend of the artery.

abdominal aorta. The outer catheter is straight while the inner catheter is curved. The advancement or withdrawal of the inner catheter will decrease or increase the curve of the tip.

In our experience, we have found the use of a modified Seldinger wire with a flexible tip of 6 to 8 inches in length very convenient. This is commercially available with a 6 inch* tip or can be readily obtained by

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* Catalog #01-0013—B & D.

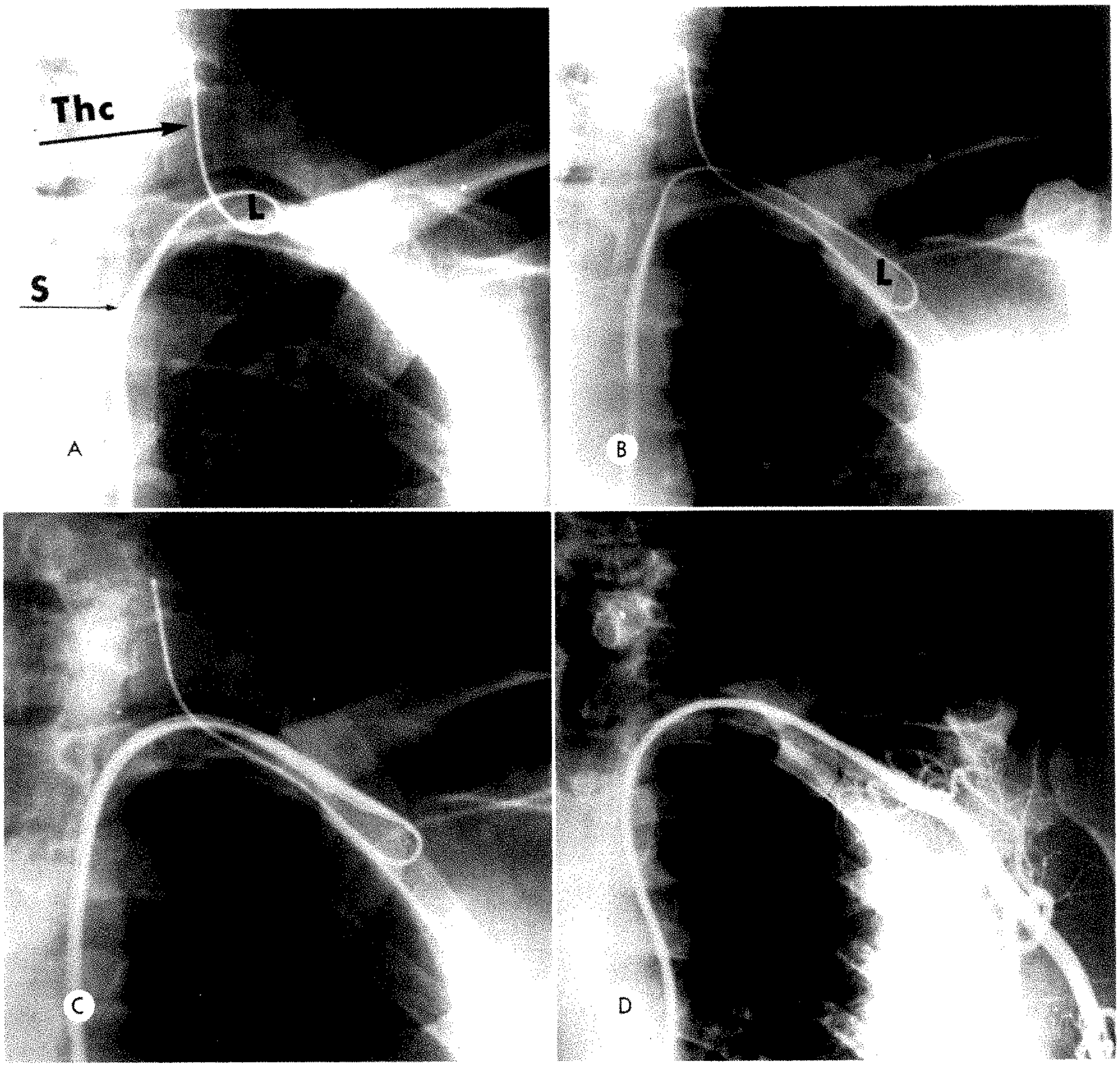

FIG. 3. Transfemoral catheterization of the left axillary artery. The use of the floppy wire is illustrated in bypassing obstacles caused by the orifices of small arteries at the bend of the artery. (*A* and *B*) Catheter in the subclavian artery (S). The tip of the wire is within the thyrocervical trunk (Thc). Note the formation of the loop (L) in the flexible part of the wire and its extension into the subclavian artery lumen. (*C*) The catheter is advanced over the wire. (*D*) Upon withdrawal of the wire, a small injection of contrast material is made to test the position of the catheter.

partial withdrawal of the removable inner core of a stainless steel spring guide,* so that the flexible part is increased to the needed length. The term "floppy" has been used for this wire because of its extreme flexibility; its applications can be divided into two groups: (I) as a loop in difficult arterial or left ventricular catheterizations, and (II) as a guide in selecting the vessel to be catheterized.

TECHNIQUES

GROUP I

A. *Difficult Arterial Catheterization.* The use of a floppy wire is suggested when, during introduction of a conventional metallic wire within the artery, an obstacle is met which cannot be bypassed by gentle pressure on the wire. The conventional wire is then replaced by the floppy wire which, once its tip is at the site of obstruction, will bend somewhere between the tip and the

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* Catalog #SG-35m—U.S. Catheter.

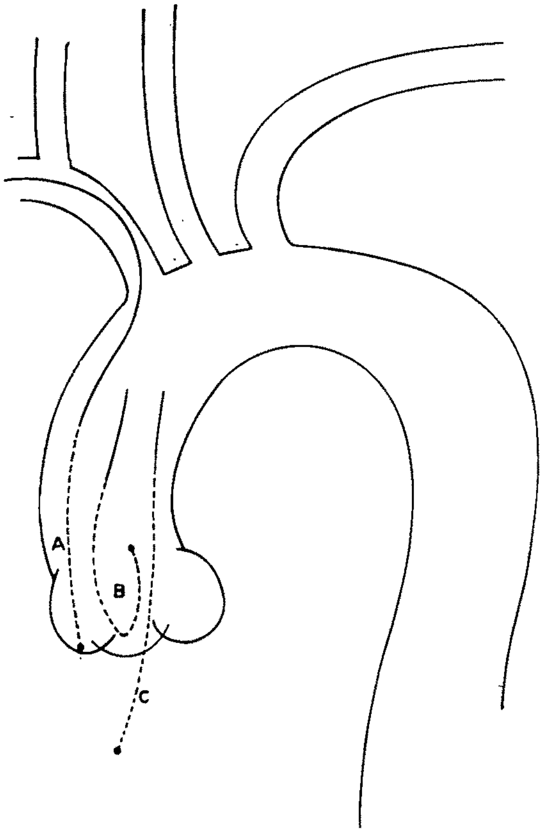

Fig. 4. Schematic drawing illustrating the passage through the aortic valve with the use of the floppy wire. The steps in the formation of the loop in the flexible part of the wire and its eventual entry into the left ventricle are designated by A, B and C.

junction with the stiff part (Fig. 2*A*). If further pressure is applied on the wire, the initial bend will result in a complete loop of the flexible part while the tip is anchored at the arterial wall (Fig. 2, *B* and *C*). This loop then acts as a new tip within the lumen of the artery and may be introduced further without applying any direct pressure on the obstacle. The catheter is then advanced over the wire, which is slowly withdrawn.

We have found the floppy wire of great help in the following cases: (1) sharp curve in the course of an artery (Fig. 2*A*); (2) bend with arteriosclerotic plaque (Fig. 2*B*); and (3) small vessel arising from the convexity of the artery at the bend, for example, the thyrocervical trunk or vertebral artery (Fig. 2 *C;* and 3, *A–D*).

In cases with marked narrowing or complete obstruction of the artery, the use of the floppy wire will not be of help, and further manipulation is not recommended.

B. *Left Ventricular Catheterization.* A similar technique may be used in passing through the aortic valve when several attempts with a catheter have failed. The tip of the wire is anchored within a sinus of Valsalva and the loop which is formed will eventually enter the aortic ostium (Fig. 4, A, B and C). Care must be taken to avoid entering the coronary arteries.

GROUP II

A. *Selective Catheterization of the Brachiocephalic Trunks.* A U-shaped catheter is advanced from the axillary artery and placed within the ascending aorta. Once free within the aorta, the tip will assume its former shape and therefore point toward the orifice of the great vessel. A floppy wire is then introduced into the catheter until it extends far beyond the catheter tip (Fig. 5*A*). Due to the flexibility of the wire, the catheter shape is not disturbed and the tip of the wire will point in the same direction as the catheter. By further advancement of the wire and combined manipulation, the tip will enter the desired artery arising from the aortic arch (Fig. 5*B*). The position of the wire indicates the artery entered, and once the wire is in place, the catheter can easily be introduced by following the leading wire (Fig. 5*C*).

B. *Selective Hypogastric Artery Catheterization.* A moderately soft radiopaque catheter with a U-shaped curve is advanced from the femoral artery into the abdominal aorta (Fig. 6*A*, 1). The catheter, straightened by its passage into the aorta, is made to resume its shape by engaging the tip in one of the orifices of a small branch of the abdominal aorta and by further advancement. The tip of the catheter then points downward to the aortic bifurcation (Fig. 6*A*, 2).

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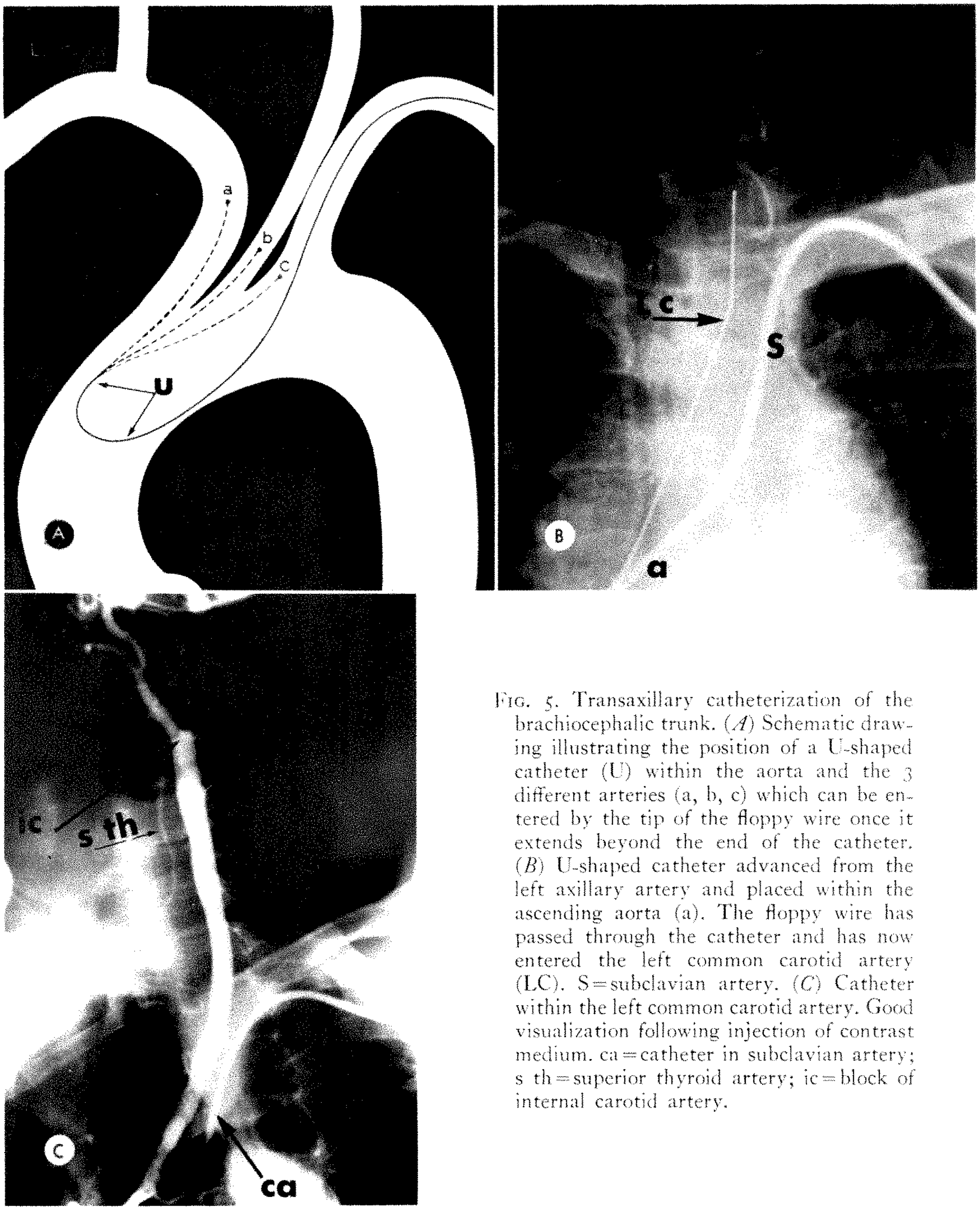


Fig. 5. Transaxillary catheterization of the brachiocephalic trunk. (*A*) Schematic drawing illustrating the position of a U-shaped catheter (U) within the aorta and the 3 different arteries (a, b, c) which can be entered by the tip of the floppy wire once it extends beyond the end of the catheter. (*B*) U-shaped catheter advanced from the left axillary artery and placed within the ascending aorta (a). The floppy wire has passed through the catheter and has now entered the left common carotid artery (LC). S = subclavian artery. (*C*) Catheter within the left common carotid artery. Good visualization following injection of contrast medium. ca = catheter in subclavian artery; s th = superior thyroid artery; ic = block of internal carotid artery.

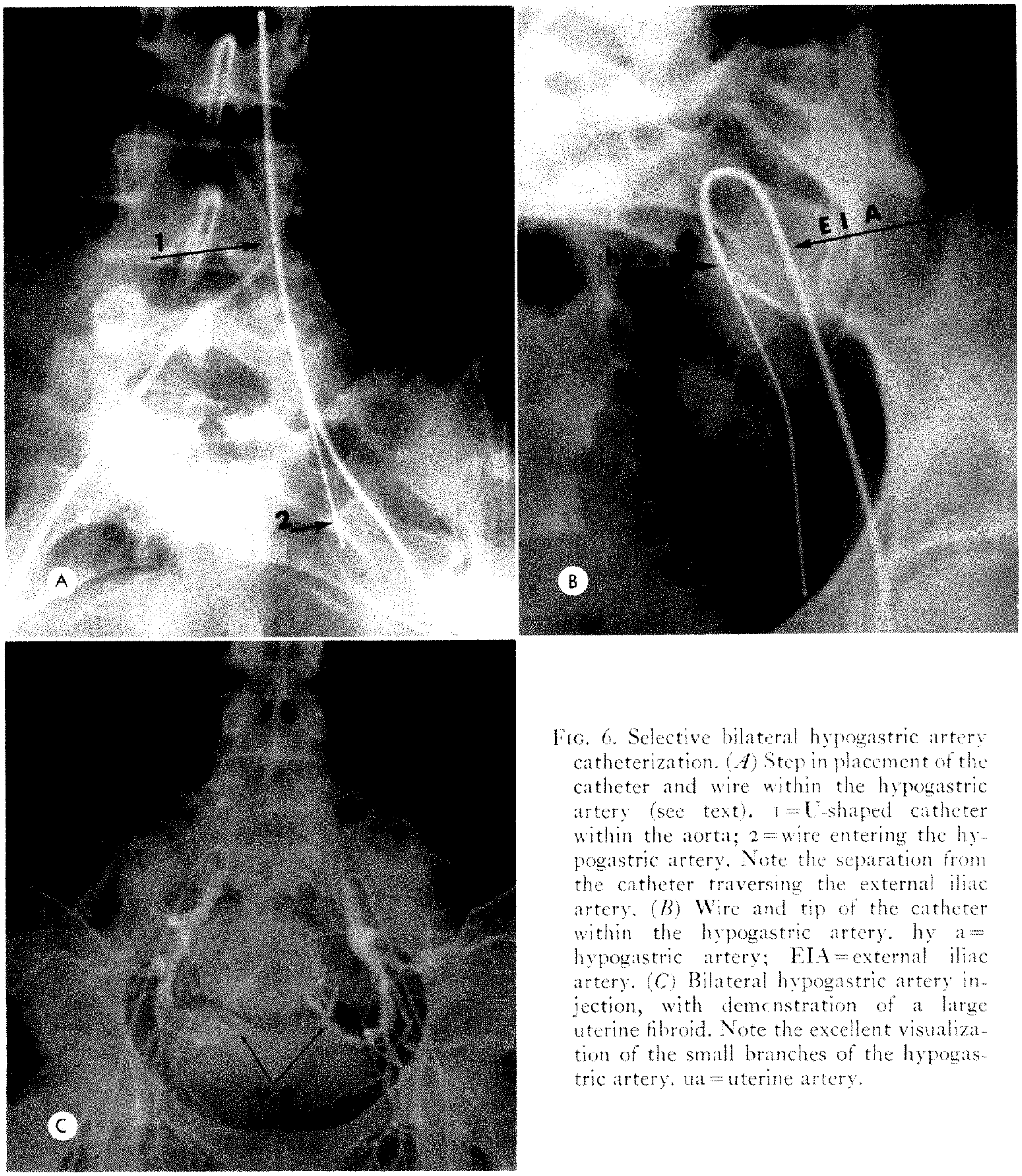
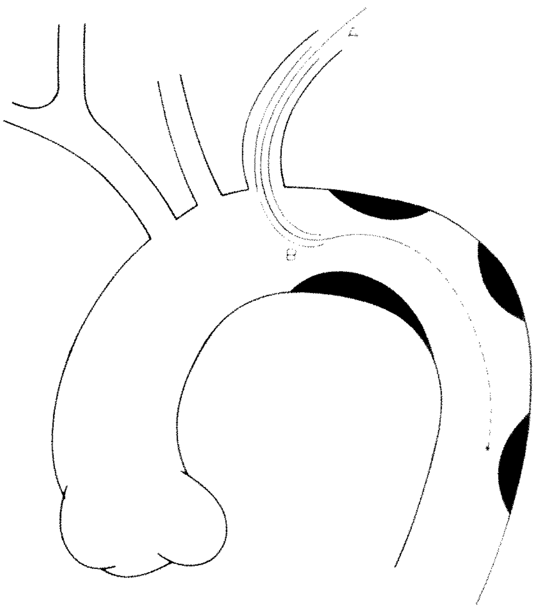


Fig. 6. Selective bilateral hypogastric artery catheterization. (*A*) Step in placement of the catheter and wire within the hypogastric artery (see text). 1 = U-shaped catheter within the aorta; 2 = wire entering the hypogastric artery. Note the separation from the catheter traversing the external iliac artery. (*B*) Wire and tip of the catheter within the hypogastric artery. hy a = hypogastric artery; EIA = external iliac artery. (*C*) Bilateral hypogastric artery injection, with demonstration of a large uterine fibroid. Note the excellent visualization of the small branches of the hypogastric artery. ua = uterine artery.

Fig. 7. Schematic drawing demonstrating the use of the floppy wire in transaxillary catheterization to direct the tip of the catheter into the descending aorta, when marked arteriosclerosis and elongation are present. A c-shaped catheter (B) is advanced from the left axillary artery into the aorta. The tip of the catheter is made to point toward the descending portion of the aortic arch. A floppy wire (A) is then advanced through the catheter into the descending aorta for several centimeters. The catheter is then advanced over the wire.



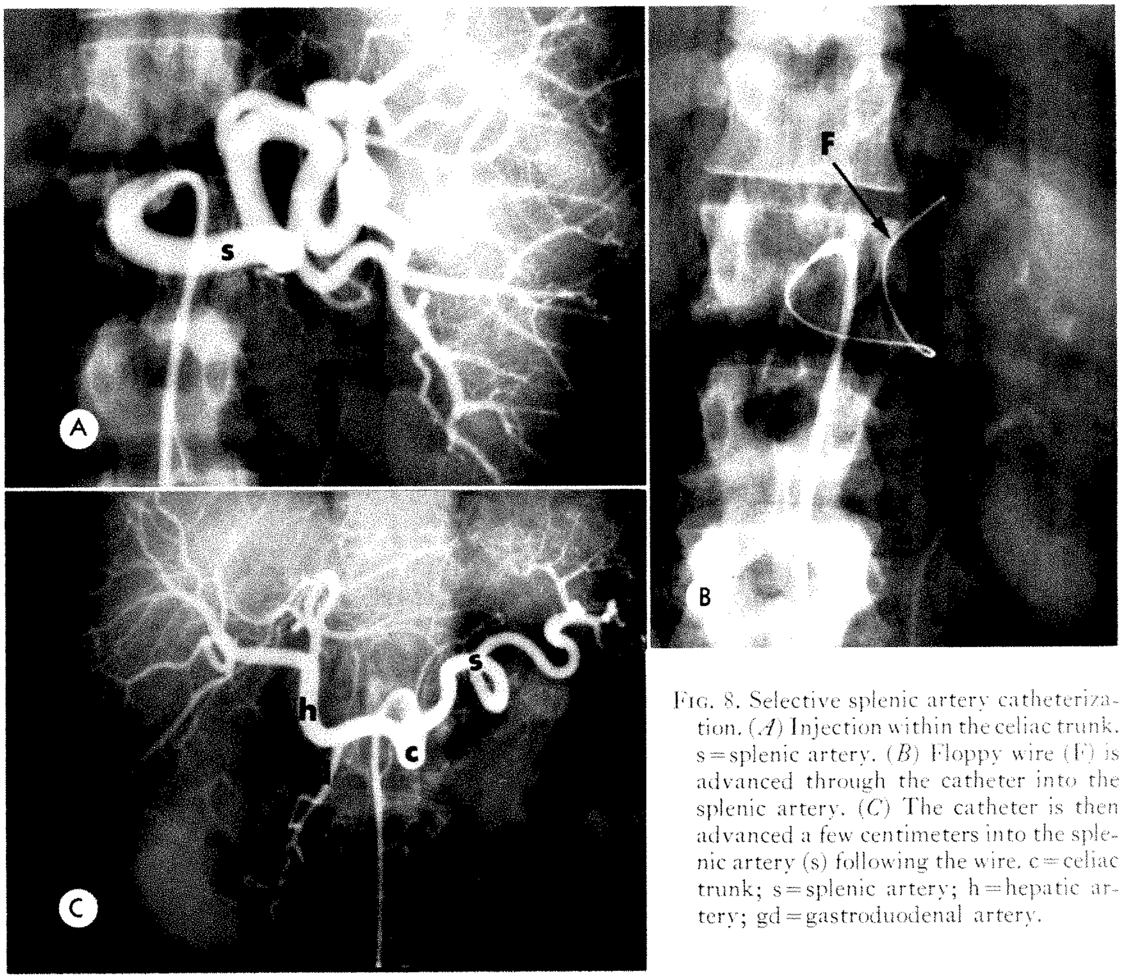


Fig. 8. Selective splenic artery catheterization. (*A*) Injection within the celiac trunk. s = splenic artery. (*B*) Floppy wire (F) is advanced through the catheter into the splenic artery. (*C*) The catheter is then advanced a few centimeters into the splenic artery (s) following the wire. c = celiac trunk; s = splenic artery; h = hepatic artery; gd = gastroduodenal artery.

The floppy wire is advanced through the catheter and its tip is made to extend far beyond the catheter end. As in catheterization of the brachiocephalic vessels, the extreme flexibility of the wire avoids change in the curve of the catheter. By combined manipulation of the catheter and wire, the tip of the wire is placed, first, within the iliac artery, and, subsequently, into the hypogastric artery (Fig. 6*B*). The catheter is then slipped in without difficulty over the wire and the wire is withdrawn (Fig. 6*C*).

C. *Other Uses*. Other uses of the floppy wire, catheterization of the descending aorta in arteriosclerotic cases and selective catheterization of the splenic artery, once the tip of the catheter is within the celiac trunk, are shown in Figure 7; and 8, *A*, *B* and *C*.

CONCLUSION

In over 350 cases, the use of a floppy wire has proven to be of considerable help in overcoming difficulties commonly encountered in arterial catheterization. No complications have followed the use of this special wire in difficult or finely selective arterial catheterization. We feel that this is an extremely valuable tool and should be available when arterial catheterization is attempted.

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A NEW METHOD OF PERCUTANEOUS CATHETERIZATION*

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THE percutaneous method of vascular catheterization has enabled the radiologist to perform a great variety of arterial and venous angiographies with relative ease and safety. However, this method has not been widely accepted for cardiac catheterization and angiocardiography, despite the obvious advantage of avoiding a cut-down, arteriotomy, and arterial suturing. The conventional percutaneous technique requires a catheter with an end hole and a tip tapered to fit a guide wire. Because of these necessary modifications, the catheter type is quite limited.

A wide variety of catheters has been developed for cardiac catheterization which can only be introduced by the cut-down method. Some have been designed for performing hydrogen and dye solution studies and for recording intracardiac electrocardiograms, intracardiac phonocardiograms, and other physiologic measurements. Others with flexible solid tips have been especially useful in crossing the aortic valve. The best angiocardiographic catheters have a closed end with multiple side holes. These catheters are safer for angiocardiography than the conventional end-hole percutaneous catheters for high pressure injections into the heart because they are less likely to recoil, and myocardial perforations are not as apt to occur.[1,3] Since these special catheters for physiologic and angiographic study of the heart must be introduced by the cut-down method, it seemed that if a way could be found to insert them percutaneously, the range and flexibility of the percutaneous technique would be greatly enhanced. Many radiologists are familiar with the percutaneous methods and are expert in their use, while few have developed the ability to suture an artery with minimal risk.

The solution to this problem would be a thin-walled sheath introduced into an artery or vein over the conventional percutaneous catheter which could be inserted in the usual fashion. This sheath would act as an extension of the vessel out of the body, and catheters or other instruments could then be introduced through the sheath into the vascular system.

Although the idea is simple, the material demands for such a sheath are great. The ideal sheath should be very thin-walled so that the puncture hole in the artery would not be significantly enlarged. With enlargement of the puncture hole, there would be an increased risk of complications. Too much variance in the diameters of the sheath and catheter would cause bleeding around the catheter when the sheath was withdrawn. A low coefficient of friction is desirable so that exchange catheters will slide readily through the sheath. Some degree of malleability of the sheath is important to accommodate slight discrepancies in catheter diameter. The sheath must be smooth and inert so that it will not injure the blood vessel.

To date, we have been unable to find a substance that completely satisfies the above requirements. Teflon* showed promise but failed because it could not be obtained thin enough and strong enough for arterial studies, though it was satisfactory for venous studies where the wall thickness of the sheath was less critical. Experiments

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* Teflon is Du Pont's registered trademark for its brand of polytetrafluoroethylene.

* Presented at the Sixty-sixth Annual Meeting of the American Roentgen Ray Society, Washington, D.C., September 28–October 1, 1965.
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with various soft metals for this purpose have been unsatisfactory.

Originally, we considered mylar,* which is extremely tough and strong, but found that it could not be extruded. Further investigation revealed a source for mylar tubes† which were being manufactured for insulating electric wire. These tubes are not extruded but are wrapped and bonded with an adhesive, very similar in construction to a drinking straw. This material, while not ideal, is far superior for the intravascular sheath to anything else that has been found. The sheath is very strong with a wall thickness of only .002 inches. Its main disadvantage is that it is rigid and will not stretch to accommodate imperfect catheter diameter tolerances. However, the component parts of the sheath system can be made uniform, overcoming this problem.

The sheath percutaneous system consists of three component parts: (1) the introducing teflon catheter necessary to insert the sheath into the vessel (a teflon catheter is superior for this purpose because of its low coefficient of friction), (2) the sheath itself, and (3) the catheter or catheters that are to be used for the study. The tolerances of the diameters of these component parts are quite critical. The sheath must fit snugly over the introducing teflon catheter. If the sheath is too large, it will bind on the subcutaneous tissue and will not follow the catheter into the vessel. The cardiac catheter introduced into the sheath must be small enough to slide freely and large enough to prevent leakage of blood around the catheter. The diameters of commercially available catheters vary considerably. It is possible to use a gauge and to reject the catheters that are too large. However, by this method, most of the catheters would be rejected.

In order to match the three components of the sheath system, we obtained mylar tubing in sizes .003 inches greater than the desired catheter French size, *i.e.*, for a No. 6 French catheter, .069 inches outer diameter, the inner diameter of the mylar sheath would be .072 inches. The wall thickness of the mylar is .002 inches so that the outer diameter of the sheath is .076 inches. The commercially supplied No. 6 French teflon catheters, which are used as the introducing catheters, tend to be more uniform than other cardiac catheters, and they usually fit snugly within the sheath without modification. However, we can make our own teflon catheters to size by using a catheter former.‡ The main problem lies with the commercially available, closed-end cardiac catheters which vary in their outside diameter considerably. We first gauge the catheters using an appropriate-sized hole in a brass block to ensure uniform catheter diameter. We can reduce the diameter of the catheters which are too large by a few thousandths of an inch by warming the catheter in boiling water and pulling it through the gauge hole. The catheter diameter can be reduced by this method without harming the catheter.

The sheath is made as short as possible in order to reduce friction. Most sheaths are approximately 7 cm. long. We usually make the sheath somewhat longer than this originally and trim it to the desired length at the time of the procedure when the size of the patient is known. A piece of cylindrical steel is glued§ to the sheath for a handle. One-half cm. of the mylar sheath protrudes beyond the steel handle. The flow of arterial blood is stopped when the sheath is introduced into the artery by crimping this end of the sheath with the fingers.

The technique of introducing the sheath into the vessel has been described previously.[2] The three components of the sheath-catheter system are shown in Figure 1. A Lehman ventriculography, closed-end, angiographic catheter is shown at the top of this figure with the mylar sheath in the middle and the teflon introducing percutaneous catheter at the bottom.

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* Mylar is Du Pont's registered trademark for its brand of polyester film.

† Stone Paper Tube Company (Division of Stone Straw Corporation), 900–922 Franklin Street, N. E., Washington, D. C. 20017.

‡ Cook, Incorporated, 300 South Swain Avenue, Bloomington Indiana 47403.

§ Eastman 910 Adhesive (registered trademark), manufactured by Eastman Chemical Products, Inc. (Subsidiary of Eastman Kodak Company), Kingsport, Tennessee.

Following local anesthesia, a skin nick measuring approximately one-half inch is made. The incision is made deep enough to entirely incise the dermis to allow free passage of the sheath through the skin. The artery or vein is punctured in the usual manner, the teflon introducing catheter is passed into the vessel, and the guide wire removed (Fig. 2). The catheter is wiped with heparin, which serves as a lubricant and prevents coagulated blood from binding the sheath. During these preliminary moves, the sheath is well back on the introducing catheter. The sheath is then advanced over the teflon catheter to the skin. The sheath and catheter are grasped together just above the incision and rotated gently into the vessel. It is important that they are held firmly so that they advance together. A little practice is required to develop the feel of the sheath passing into the artery. The sheath is advanced to the steel tubing. The angiographic or other cardiac catheter is then wiped with heparin. The teflon catheter is withdrawn, and the end of the sheath is crimped with the fingers to stop the flow of blood. The cardiac catheter is then slipped through the sheath, and the sheath is withdrawn to the distal end of the cardiac catheter out of the way. The cardiac catheter is best manipulated in this fashion, although it is possible to leave the sheath within the vessel and to manipulate the catheter within the sheath. If a new catheter is desired, the initial catheter is withdrawn until only a few centimeters remain within the vessel.

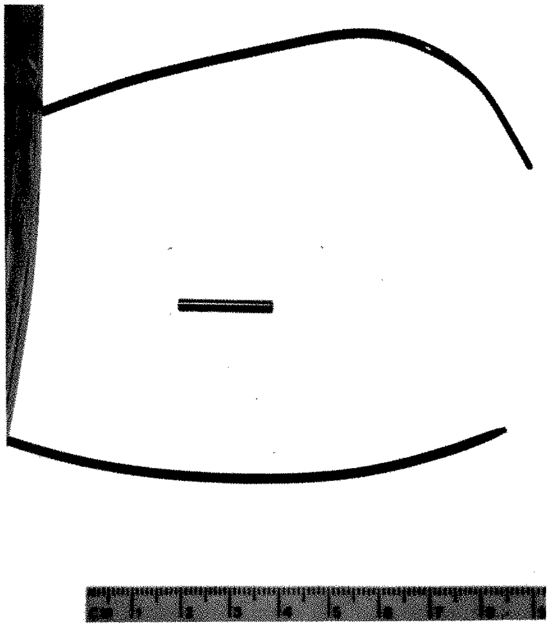

Fig. 1. Photograph of a Lehman ventriculography, closed-end, angiographic catheter (top) with the mylar sheath (middle) and the teflon introducing percutaneous catheter (bottom).

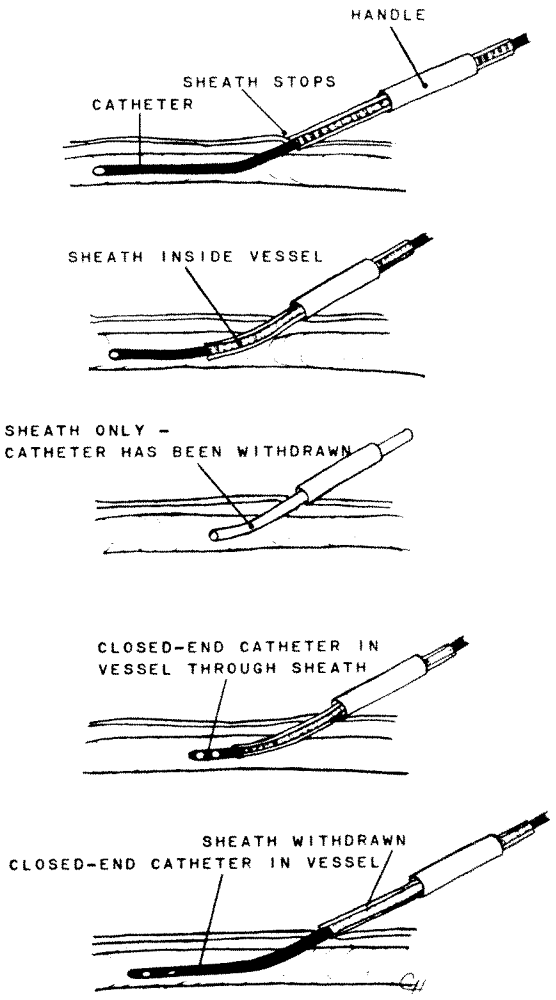
HANDLE
SHEATH STOPS
CATHETER
SHEATH INSIDE VESSEL
SHEATH ONLY – CATHETER HAS BEEN WITHDRAWN
CLOSED-END CATHETER IN VESSEL THROUGH SHEATH
SHEATH WITHDRAWN
CLOSED-END CATHETER IN VESSEL

Fig. 2. Illustration of the method of catheter exchange following introduction of an open-end catheter into the vessel. (Reproduced with permission from *Radiology*.[2])

The mylar sheath is then advanced to the skin and reinserted into the artery in the manner described above. This catheter is then removed and the exchange catheter inserted after first being wiped with heparin.

RESULTS

We have used this system almost exclusively in children ages 2 to 16 years. Most of our cases are under 12 years of age. We have used No. 5 and No. 6 French systems on these patients depending on their size. Thirty arterial catheterizations and 10 venous catheterizations were performed using the sheath. In this group, there were only 2 failures because of the sheath. On one occasion, after successfully introducing the Lehman ventriculography catheter into the aorta through the mylar sheath, the catheter could not be passed into the left ventricle. A second catheter was prepared with a different curve on the tip but could not be exchanged because the original catheter became wedged within the sheath and could not be withdrawn. In the second case, the sheath could not be introduced into the artery. This was the only occasion in which this difficulty occurred. The patient had a previous catheterization in the right groin by the cut-down method with a lot of fibrous tissue. The sheath could not be passed through this tissue into the artery.

To date, no significant complications have occurred. One 12 year old girl had a transient marked diminution in pulses for 12 hours, which was attributed to arterial spasm.

With a system of matched components, passage of the sheath into the artery and subsequent introduction of the closed-end catheter can be performed without difficulty. Occasionally, some difficulty is encountered in exchanging catheters. Some of the catheters tend to expand slightly after being warmed by the blood stream. On occasion, it has been difficult to withdraw a catheter from the sheath or to exchange a new one. This problem is not encountered if teflon catheters are used because of the extremely low friction coefficient of teflon. Also, with our system of matched catheters, this has become much less of a problem. Because the mylar is wrapped like a drinking straw, severe twisting of the sheath can result in reducing the inner diameter. To exchange catheters, the original catheter should be withdrawn almost completely from the vessel and wiped with heparin before the sheath is reinserted. The sheath should then be withdrawn slightly from the vessel and grasped lightly just proximal to the steel hub when the catheter is being withdrawn. As before, the end is crimped to stop the flow of blood and another catheter reinserted through the sheath. As with any procedure of this nature, careful attention to small details is essential.

SUMMARY

A method has been described whereby a closed-end catheter or other special-tipped catheter can be passed into an artery or vein percutaneously using a thin sheath of mylar. To date, the method has been used almost exclusively in children with No. 5 and No. 6 French catheters. Thirty arterial catheterizations and 16 venous catheterizations have been performed with no serious complications. In our laboratory, this method has added considerably to the flexibility and use of the conventional percutaneous method of catheterization.

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SIMPLIFIED SUBTRACTION*

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THE subtraction technique has proved to be a valuable adjunct in angiography where the initial study is inconclusive because of bone structures overlapping and obscuring the contrast filled vessels. This is especially true when the contrast medium in the vessel of interest is dilute or the overlapping bone is underpenetrated. Ziedses des Plantes[6] described the subtraction technique in 1934. Since then, he has described its many clinical applications.[7] It has not been utilized as often as it should have been because of the time-consuming, trial-and-error methods usually employed. The purpose of the authors is to describe a simple, quick and reproducible method of subtraction that has yielded uniformly good results. It requires no special equipment, merely that which is available in any radiology department.

MATERIALS

The materials necessary for this method include the following:

1. An ordinary radiographic cassette with high-speed screens.
2. Par-speed, fine-grain, double-emulsion, radiographic film.
3. High-speed radiographic film.
4. Black matte paper such as the interleaving paper included in cartons of radiographic film.
5. An ordinary viewbox used in the horizontal position.
6. A simple contact printer. An ideal printer can be constructed by replacing the back of a 14×17 inch cassette with a $\frac{1}{8}$ inch plate glass that is hinged with adhesive tape at one end. The back of the front screen is covered with the black interleaving paper.
7. A 60 watt incandescent light bulb.

TECHNIQUE

The following roentgenograms are used in the subtraction technique: A preliminary "scout" film of the area of interest and an angiogram with the vessels in that area opacified, called the "contrast" film. No movement of the patient or tube between exposure of the scout film and the contrast film should occur. When a serial changer is used, an early film, *i.e.*, without contrast medium in the area of interest, may be used as the scout film. This is usually superior to a capillary or venous phase film which can also be used as the scout film because movement of the patient is more likely to have occurred in the later (capillary or venous) phase of the study. Our technique consists of two stages (Fig. 1).

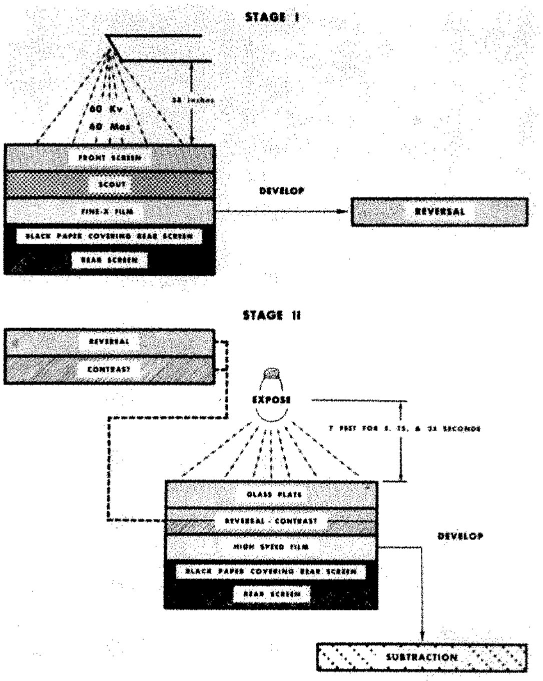

FIG. 1. Diagrammatic representation of the stages and steps of the subtraction technique.

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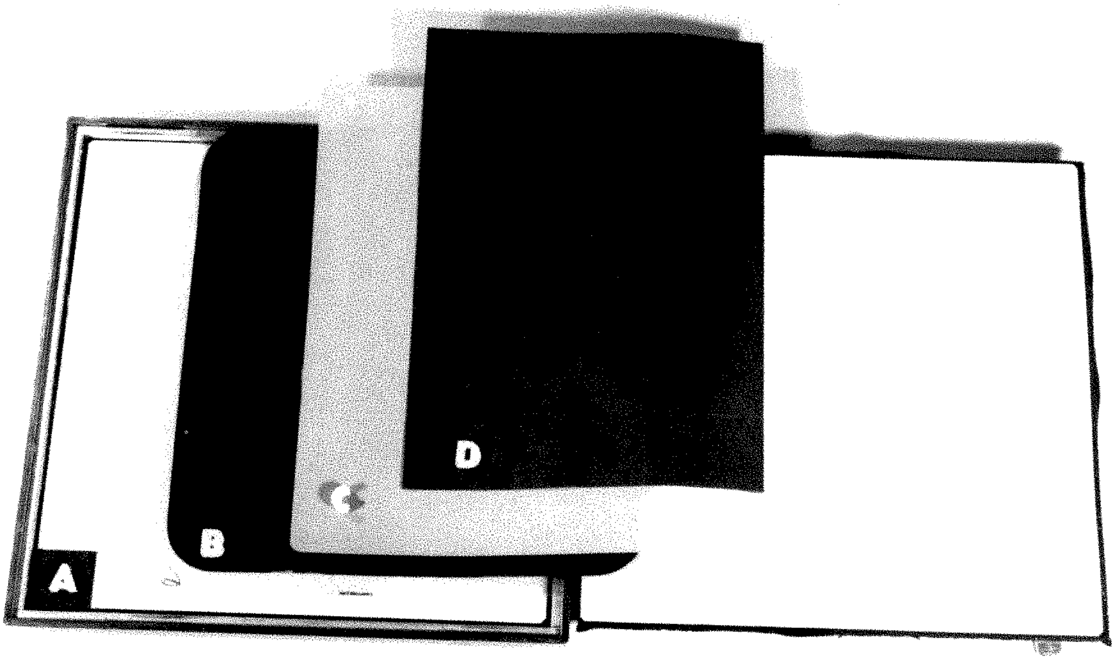

Fig. 2. Photofluorographic reversal of the scout film. The arrangement or "sandwich" in the cassette consists of (A) the front screen, (B) the scout film, (C) a sheet of par-speed, fine-grain radiographic film, and (D) a sheet of black matte paper covering the front of the rear screen.

STAGE I. THE REVERSAL PROCEDURE

Step 1. A photofluorographic reversal of the scout film is obtained by using the light of the front high-speed screen of the ordinary radiographic cassette. The arrangement or "sandwich" in the cassette shown in Figure 2 consists of (A) the front screen, (B) the scout film, (C) a sheet of par-speed, fine-grain radiographic film, and (D) a sheet of black matte paper covering the front of the rear screen. The cassette, loaded in the dark, is exposed to roentgen rays with factors of 60 kvp., 60 mas., and a 38 inch focal film distance. Filters, *i.e.*, the 2½ mm. aluminum filtration which includes the inherent tube filtration present in the ordinary overhead roentgen-ray tube, are not removed.

Step 2. The exposed undeveloped film is then processed in an automatic unit or in a manual tank with the usual processing time and temperature. On the resulting film, the normally white structures are black, and *vice-versa*. It is therefore called the "reversal" film.

STAGE II. THE SUBTRACTION PROCEDURE

Step 1. The reversal film is exactly superimposed upon the contrast film with the resultant "graying out" of the bone structures. The contrast filled vessels will appear as a white shadow on a gray background. This is most easily accomplished on a horizontal viewbox in a darkened room. The films are taped together in this position with clear cellophane tape. Masking the edges of the viewbox with blackened roentgenographic film blocks out excess light and thus facilitates exact superimposition. If exact superimposition of all the areas is unobtainable, one should at least superimpose the area of major interest.

Step 2. The final subtraction film is a contact print made in the darkroom with the light of a 60 watt bulb at a distance of 7 feet. The contact printer (cassette) is used with the following sandwich arrangement from front to back: (A) glass plate, (B) the combined reversal-contrast film, (C) an unexposed high-speed radiographic film, and (D) the black matte interleaving paper

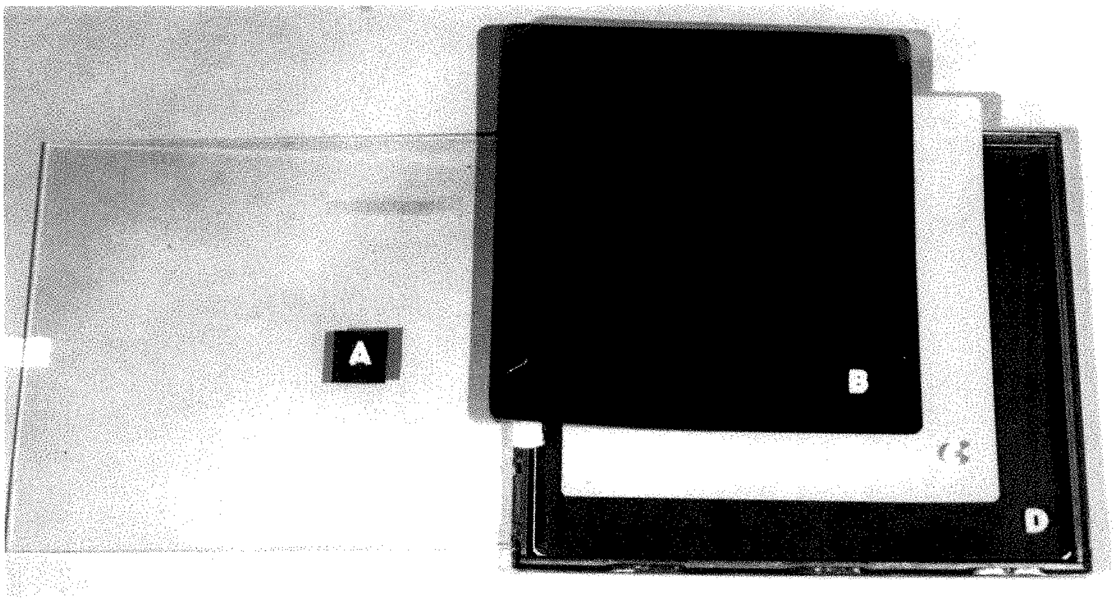

Fig. 3. Contact printing of the subtraction film. The arrangement or "sandwich" in the contact printer (cassette) consists from front to back of (A) the glass plate, (B) the combined reversal-contrast film, (C) an unexposed high-speed radiographic film, and (D) the black matte paper covering the front of the rear screen.

(Fig. 3). The printer is exposed through its glass front to the light of the 60 watt bulb for 5 seconds. The high-speed film (subtraction film) is then removed and processed in an automatic or manual processing unit at standard technique. This procedure is repeated using 15 and 25 second exposures. These three exposures always ensure that at least 1 (and usually all) of the subtraction films is of diagnostic quality. If the reader decides to make only a single exposure, the 15 second film has proven to be the exposure of choice.

DISCUSSION

We have used the photofluorographic method for obtaining the reversal film (Stage I) and the contact printing method for the subtraction film (Stage II) as did Levick and Mitchell.[3] We feel that the photofluorographic method, using a fine-grain double emulsion par-speed film, yields a reversal with an optimum range of densities. The phrase "optimum range of densities" means that the mid-range grays are present on the reversed film. Some authors[1,2] have recommended contact printing for both the reversal and subtraction film. The abnormal range of density (a lack of mid-range grays) on a contact-printed reversal film does not match the normal range of grays present on the angiogram and, therefore, complete subtraction is impossible. The double emulsion film records detail adequately so that special single emulsion film, as recommended by others,[2] has not been required. Using the contact printing method in the reversal stage has the additional disadvantage of very short and, therefore, critical light exposure times. As described by Rockoff and Camp,[4] this exposure time depends upon several variables and is obtained only by trial and error. With our method, long, easily reproducible, noncritical exposure times can be employed. Using the contact printing method in the subtraction stage has the additional advantage of eliminating the excess fogging which sometimes is a problem with photofluorographic subtraction, as mentioned by Vezina and McRae.[5]

Complicated developing methods have

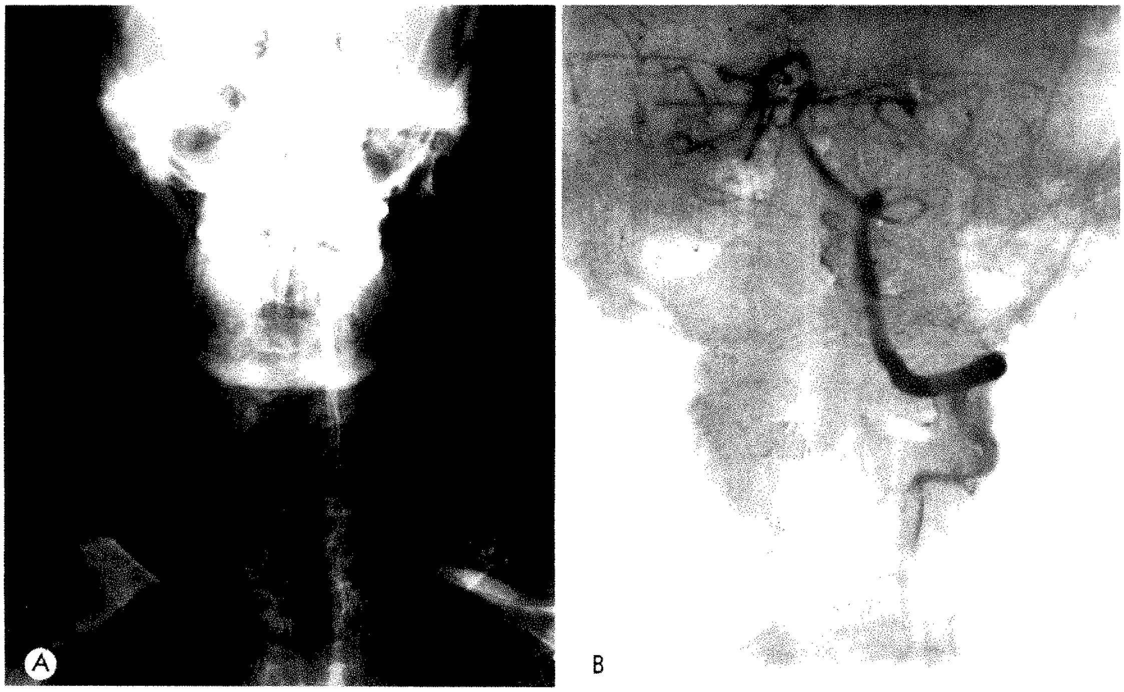

Fig. 4. (*A*) Left vertebral angiogram. (*B*) Vertebral and basilar artery detail after subtraction.

been another reason for the limited use of the subtraction technique. For example, Horenstein *et al.*[2] have described a technique in which the use of very critical manual developing times with fresh developer at a temperature of 20±0.5°C. is essential for satisfactory results. No special developing techniques are required by our method and films may be processed manually or automatically.

Several case examples (Fig. 4 through 7) are presented showing the value of subtraction and the quality of the films obtained by our technique.

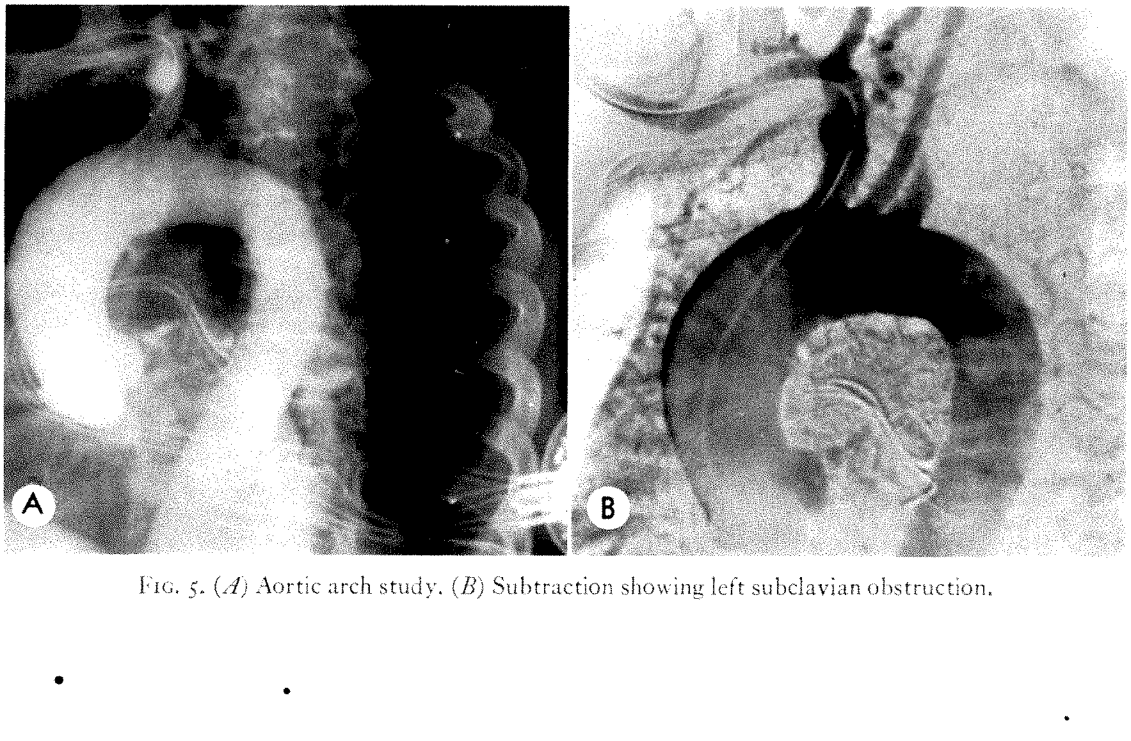

Fig. 5. (*A*) Aortic arch study. (*B*) Subtraction showing left subclavian obstruction.

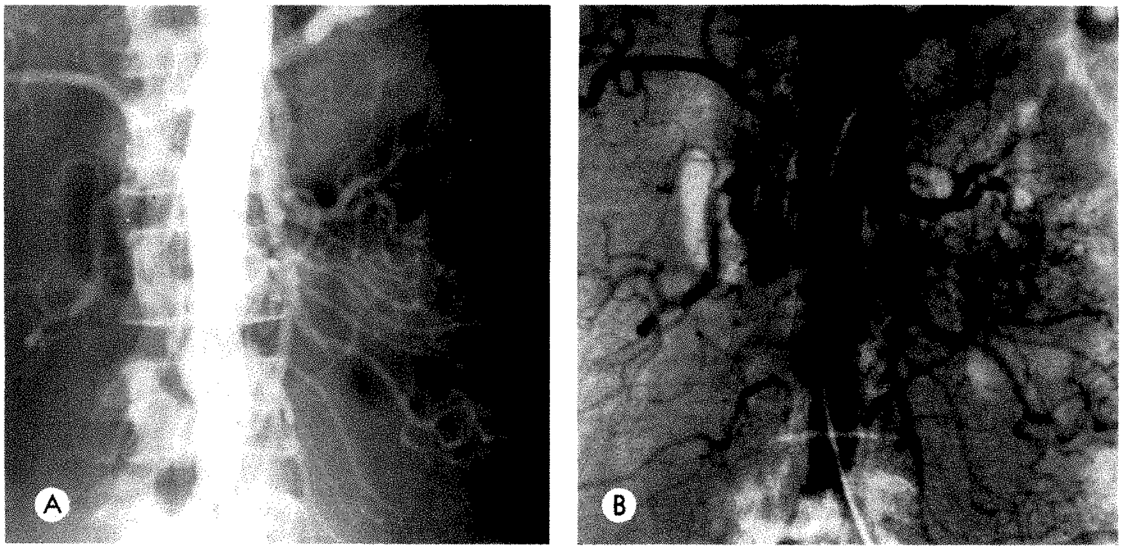

Fig. 6. (*A*) Retrograde aortogram for right aortico-renal graft. (*B*) Subtraction showing complete thrombosis of graft.

SUMMARY

We have described a simple, rapid, and reproducible method for performing subtraction. The subtraction films which result are of uniformly diagnostic quality. We have eliminated the complicated developing methods, critical exposure times and the trial-and-error aspects of the technique which have previously deterred the widespread use of subtraction. Our technique

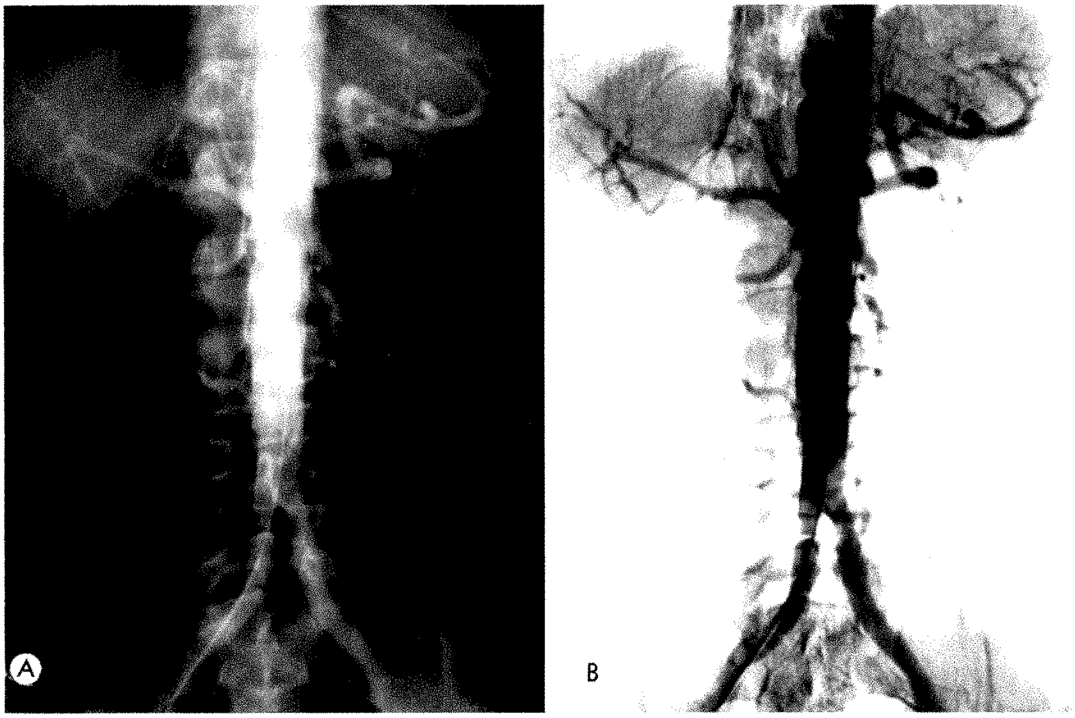

Fig. 7. (*A*) Retrograde aortogram for hypertension evaluation. (*B*) Subtraction demonstrates right renal artery stenosis.

has yielded diagnostic results from otherwise equivocal or nondiagnostic films.

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A SIMPLIFIED SUBTRACTION TECHNIQUE*

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THE principle of photographic or electronic "subtraction" of noncontributory and confusing roentgen images is well known to radiologists. A variety of relatively simple techniques has been described in the recent radiologic literature and this has resulted in some resurgence of interest in this procedure.[1,3,4] The practicality of electronic subtraction as described by Holman and Bullard[2] has given further impetus to this diagnostic technique. Although it seems obvious that television subtraction is highly desirable, it is equally obvious that the equipment necessary for this subtraction technique is not available in most radiology departments. At the present time, the simplest practical photographic subtraction technique would seem to have the widest applicability.

Our own attempts at reproduction of a satisfactory photographic subtraction technique by any of the methods described in the literature did not result in uniformly diagnostic examinations. A considerable trial and error experimentation was required with the initial use of the technique and also generally with each subsequent examination. Out of the experience, however, evolved what we consider a more simplified and more easily reproducible subtraction technique. Although quite similar to the procedure of Rockoff and Camp,[4] and of Hanafee and Stout,[1] we found that by using our modification of these techniques it was possible to establish standard exposure factors and maintain reproducible quality. It is our belief that others may easily adapt this technique to their own departments with few, if any, modifications. During the past 2½ years, we have employed this subtraction technique upon selected angiographic examinations and feel that it is of significant value in a variety of circumstances, several of which are illustrated (Fig. 1 through 7).

Basically, the subtraction technique employs the superimposition of a reversal print of a control or "scout" roentgenogram over the roentgenogram of the same area made after the injection of contrast medium. In principle and in fact, when these two films are superimposed, the blacks and whites of the reversed images tend to cancel out the detail of everything except the contrast medium. This procedure has its greatest value in angiography when overlying osseous structures obscure some critical vascular detail. Although we would not like to imply that the subtraction technique is a substitute for quality angiography, we

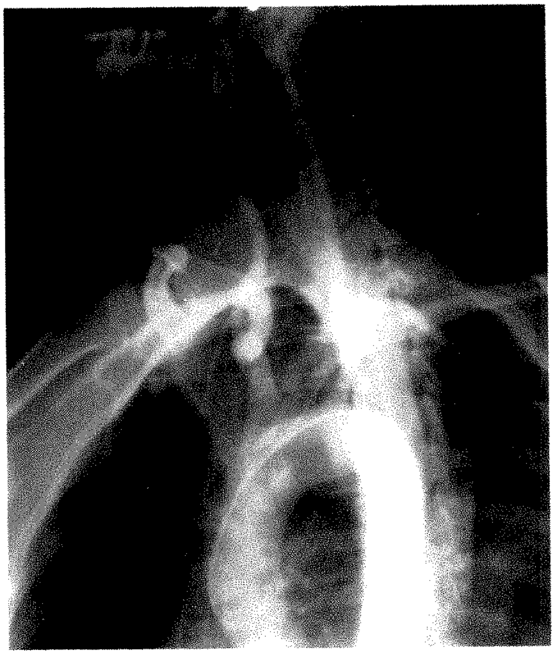

Fig. 1. Aortic arch injection in a 40 year old woman with history of two cerebrovascular accidents. Origin of left carotid artery not well shown.

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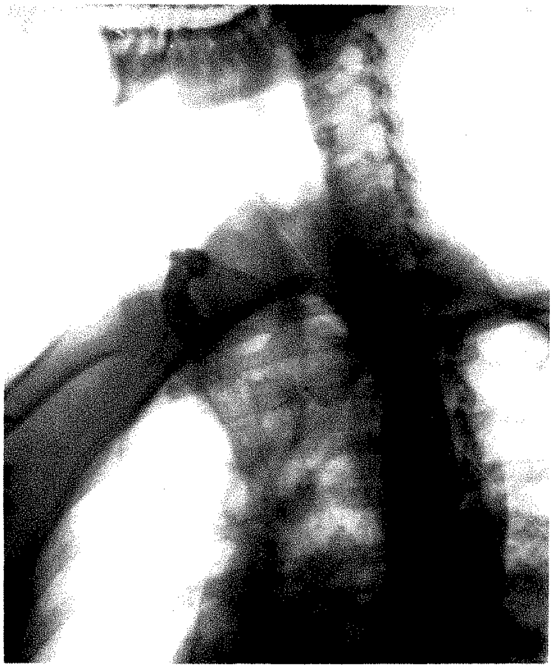

Fig. 2. Same patient as in Figure 1. Reversal print of delayed film exposed at end of series (no contrast medium).

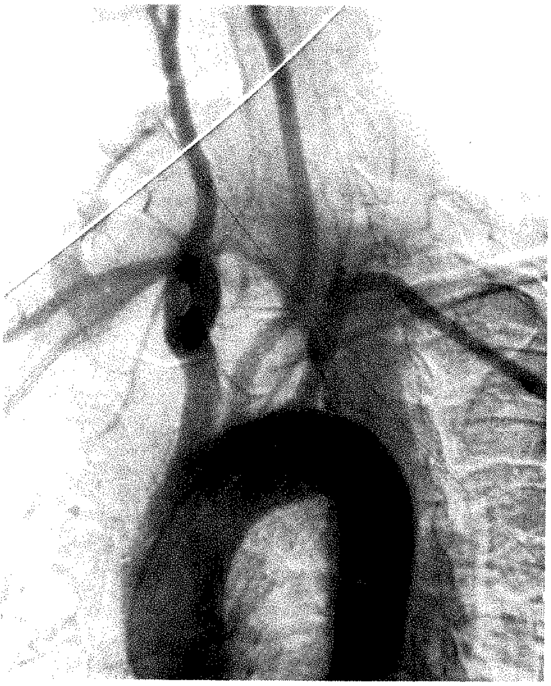

Fig. 3. Same patient as in Figures 1 and 2. Subtraction film obtained by superimposition of Figure 1 and Figure 2 shows normal origin of left carotid as well as better definition of all arch vessels.

have salvaged some examinations of otherwise limited value.

Our own technique is based on several points suggested in the literature and confirmed by our own experience. We quickly learned that incandescent light provided a more constantly reproducible reversal print than did some of the roentgenographic manipulations which have been described. The use of a 15 Watt light bulb at a 6 foot distance, as suggested by Rockoff and Camp,[4] enabled the use of an exposure time of 1 second or greater. This made possible the use of a relatively inexpensive dark room timing device. We use Kodak commercial film which is a single emulsion film that can be automatically processed. Hanafee and Stout[1] also suggested the use of this film, but employed a hand developing technique.

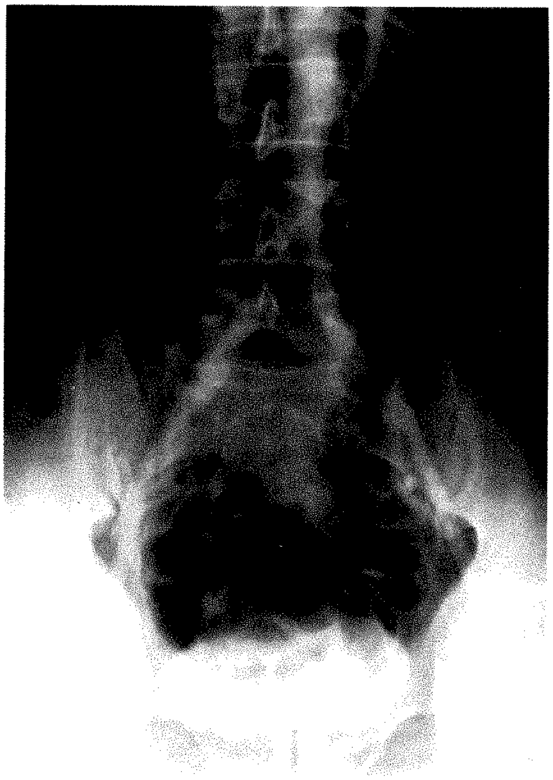

Fig. 4. Best film of translumbar aortogram series in patient with occlusive vascular disease of lower extremities. Origin of right common iliac not well shown and possibly narrowed.

The cassette used in our subtraction technique was handmade by replacing the front panel and screen of a 14×17 inch cassette with window glass of the same size. The rear intensifying screen was covered with a thick cardboard filler.

Since the subtraction technique depends upon having a perfectly matched control film, we have established the routine of including a delayed exposure film in virtually every serially filmed arteriographic series. This has proved much more satisfactory than a scout film obtained prior to the injection of contrast medium, because the delayed serial film is more likely to be exposed in exactly the same phase of inspiration and can be more accurately superimposed over the arteriogram.

DETAILS OF TECHNIQUE

A sheet of the Kodak commercial film is loaded in the glass front cassette with the nonemulsion (glossy side) facing the rear of the cassette. The previously described delayed exposure film of the arteriographic series (film without contrast medium) is placed on the commercial film and the cassette glass cover closed. A 1 second exposure of the 15 Watt bulb at a 6 foot dis-

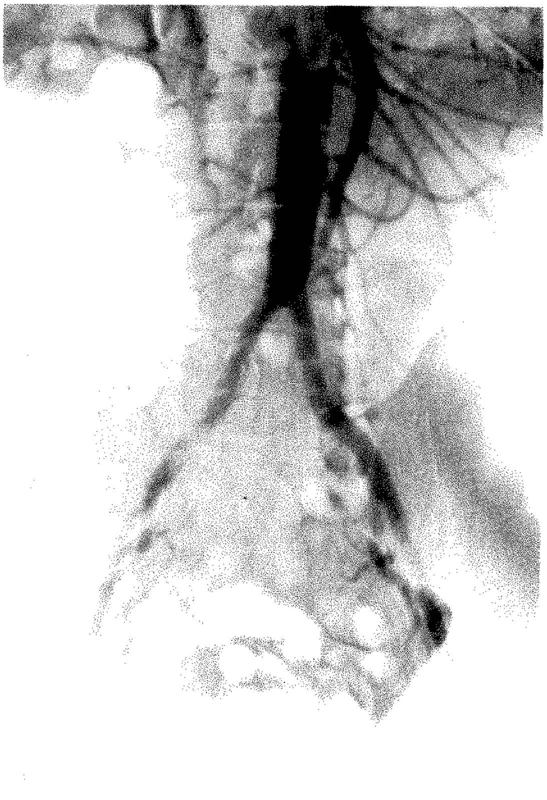

Fig. 5. Same patient as in Figure 4. Subtraction film showing only minimal atheromatous disease of both iliacs.

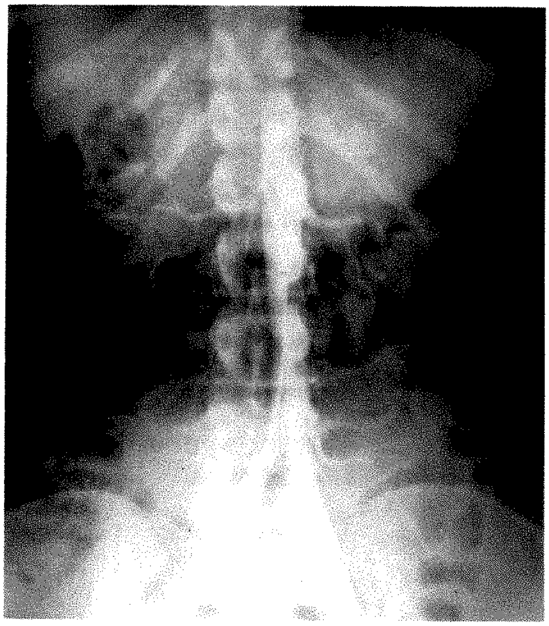

Fig. 6. Best film of suboptimal retrograde femoral aortography. Right renal artery origin poorly visualized.

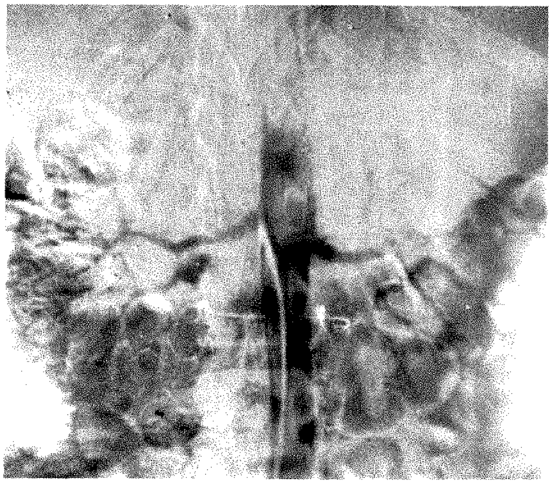

Fig. 7. Same patient as in Figure 6. Subtraction film shows normal right renal artery.

tance is made. We use a goose-neck lamp on a table and place the cassette on the floor. The exposed commercial film is then routinely processed in an automatic processor. The resultant reversal or positive print is superimposed on the angiogram to be subtracted with the films held in contact with tape at the corners. Exact superimposition is required and this aligning of the reversed prints is probably the most critical part of the entire procedure. A second commercial film is then loaded in the cassette as before and the superimposed films are placed on the commercial film and secured in position with the glass cover. A 9 second exposure is made with the 15 Watt bulb at the same 6 foot distance and this final subtraction film is processed in an automatic processor.

It is possible that the exact timing of the exposure will vary slightly, depending upon the amount of reflected illumination from darkroom walls and fixtures in other departments. In our experience, the procedure is applicable to roentgenograms of widely varying exposure techniques.

SUMMARY

A simple and easily reproducible subtraction technique is a valuable adjunct to angiography in a variety of circumstances. The procedure described can be adapted to any radiology department and is easily performed without the use of any expensive equipment.

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Representative on the Board of Chancellors of the American College of Radiology: Seymour F. Ochsner.

Sixty-seventh Annual Meeting: San Francisco-Hilton Hotel, San Francisco, Calif., September 27–30, 1966.

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President: Milton Friedman, New York, N. Y.; *President-Elect:* John L. Pool, New York, N. Y.; *1st Vice-President:* Harry Hauser, Cleveland, Ohio; *2nd Vice-President:* Daniel Catlin, New York, N. Y.; *Secretary:* Fernando G. Bloedorn, Baltimore, Md.; *Treasurer:* Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colorado 80907.

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Representative on the Board of Chancellors of the American College of Radiology: Charles G. Stetson.

Forty-ninth Annual Meeting: Royal York Hotel, Toronto, Ontario, Canada, May 29–31, 1967.

EDITORIAL

THE FORTY-EIGHTH ANNUAL MEETING OF THE AMERICAN RADIUM SOCIETY

THE AMERICAN RADIUM SOCIETY held its Forty-eighth Annual Meeting at the Camel Back Inn, Phoenix, Arizona, April 13–16, 1966. The accommodations were attractive for both the scientific and social events. The attendance by members and guests amounted to a total registration of 518, representing 148 members, 203 scientific guests and 167 ladies.

The meeting was officially opened on Wednesday morning, April 13 in the Peace Pipe Room. Dr. Justin J. Stein, President of the Society welcomed the members and guests to sunny Phoenix and indicated that the local committee wished those in attendance to take full advantage of the recreational facilities and to visit the unique sights of the surrounding country-side. Dr. Stein delivered the Presidential Address titled "Trends in the Management of the Cancer Patient."

After reviewing important events in the history of the American Radium Society, he stressed the importance of this oncological organization whose members represent various specialties and related sciences which are actively engaged in cancer diagnosis, treatment and research. The concept of team work between the various disciplines is considered essential for progress in the management of the cancer patient. The influence of the federal government in establishing regional cancer centers should be beneficial if, as the law clearly states, this type of program will not interfere with existing patterns of medical practice. The main function of these centers would be to provide specialized training, to develop and evaluate new techniques for diagnosis and treatment, to manage patients presenting specialized problems, to provide special education for the community physician and to cooperate closely with all voluntary local, state and federal agencies engaged in health activities.

Dr. Stein also spoke on the use of clinical trials in cancer therapy, management of coexisting diseases in the cancer patient, the role of the oncologist, the value of chemotherapy, the approach to preventive measures, the need for continuing medical education, the dilemma of the shortage of physicians, the advantages of staging cancer according to the TNM system, the significance of epidemiology and the role of radiation therapy in the management of the cancer patient.

The first scientific session under the chairmanship of Dr. Joseph H. Farrow consisted of presentations on the current status of the regional training program for mammography for technicians and radiologists; mucosal melanomas of head and neck; the role of aortography in the diagnosis of cancer; and experience with iridium 192 ribbons with particular reference to radical neck implants.

Dr. German Garcia of Mexico gave a statistical analysis of therapeutic integration of breast cancer by (1) detection of breast cancer in the highest possible number of women, (2) unity of oncologic criteria concerning therapeutic behavior and (3) treatment by surgery and radiotherapy by one and the same team previously trained in each of the therapeutic aspects. Dr. Florence C. H. Chu answered the question: Does prophylactic radiotherapy given for cancer of the breast predispose to metastasis? In 1962, Dao and Kovaric reported a higher incidence of ipsilateral lung and skin metastases in those patients who received postoperative radiotherapy following mastectomy than in those who had surgery alone. In a study of 674 patients who had radical mastectomy at Memorial Center, 205 of whom received prophylactic radiotherapy and 469 patients who did not, the observations of Dr. Dao and Dr. Kovaric were not substantiated by the data.

Other subjects presented in connection with breast cancer were physical and clinical parameters in the management of advanced breast cancer with irradiation alone and the history of St. Agatha relative to inadequate simple mastectomy. Miss Margaret D. Snelling of The Meyerstein Institute of Radiotherapy, Middlesex Hospital, London, and President of the British Institute of Radiology spoke on an attempt to estimate the value of postoperative irradiation of regional lymph nodes in cancer of the breast.

The latter part of the morning session included papers on the treatment of osteogenic sarcoma of the mandible; the diagnosis and treatment of metastatic brain disease with antifibrin antibody I^{131}; and malignant lesions of the nasopharynx. Dr. Adolph Zuppinger, guest speaker from the University of Bern, Switzerland, presented material on the treatment of osteogenic and soft tissue sarcomas by the use of high speed electrons. He illustrated his talk with many color slides.

During the afternoon, a sight seeing bus tour covering downtown Phoenix, Desert Botanical Gardens, Heard Museum and the residential and resort areas of the Phoenix-Scottsdale complex was participated in by the ladies.

The second scientific session was held Thursday morning under the chairmanship of Dr. Richard H. Jesse. Dr. John L. Pool of New York presented an evaluation of 44 cases of pleural mesothelioma. Other subjects discussed were the value of 2 mv. roentgen ray therapy in differentiated thyroid carcinoma; intraarterial 5-bromodeoxyuridine combined with roentgen ray therapy; and treatment of bone metastases with parathormone followed by radiophosphorus. A group of papers on radioisotope scanning included radiation-induced hepatic damage demonstrated by radioisotope scanning; the value of strontium 85 scanning in roentgen ray therapy; color scanning of bone for the diagnosis of cancer; and a description of the uses of the gamma camera. Dr. Alexander Pearlman of New York presented studies on the tumor lethal dose in Hodgkin's disease by constructing a mathematical model to indicate the probability of success of graded dose levels above and below the iso-effect curve. Dr. R. Lee Clark, Jr., Director of the M. D. Anderson Hospital in Houston, delivered an address on the present status and future trends in cancer therapy. He stated that the merging disciplines of virology, cytology, biology and immunology may hold the key to the cancer enigma, but are still in their evolution. Today's cancer patient, who cannot wait for tomorrow's cure, is dependent upon the surgeon, the radiotherapist and the chemotherapist. Dr. Clark presented statistics from his own institution showing a substantial increase in the 5 year survival rates of many types of cancer over the past 20 years due to improvement in surgical techniques and to the advent of megavoltage radiotherapy. Within this century, the total concept of adequate, effective therapy for the cancer patient has moved from the realm of a single discipline—surgery—through the specialist explosion era, to today's multidiscipline attack. The patient of today is best treated by an oncologic team with a combination of treatment modalities.

A symposium on the advantages and disadvantages of electron beam therapy was held under the chairmanship of Dr. Charles Botstein of Montefiore Hospital of New York. Dr. Adolph Zuppinger pointed out that electron beam therapy was tried over 40 years ago and in recent years has evolved into a cancer radiation modality of greater value. His experience since 1958 shows that in many instances electrons produced a better biologic effect than photons. He cited many cases of successful treatment, including carcinoma of the vulva, metastases to inguinal lymph nodes secondary to carcinoma of the penis, carcinoma of skin of the ear, mixed tumors and cylindromas of the parotid gland, papillary carcinoma of the thyroid gland, fibrosarcoma of the abdominal wall, chordoma of the spine, bronchogenic carcinoma, and abdominal wall recurrence from a gastro-intestinal tract primary carcinoma. The dosage ranged from 6,000–8,000 r in 20 to 40 days with variation of the mev. levels depending upon the depth of the lesion.

Dr. Maurice Tubiana of the Institut Gustav Roussy in France presented information on the relative biologic effectiveness of high speed electrons and the difference from cobalt 60 and other megavoltage modalities. Higher dosage of electrons will eliminate the heterogeneity of irradiation usually found in megavoltage photon beams.

Dr. John S. Laughlin of Memorial Hospital in New York also emphasized the monochromatic spectrum of high energy electrons. They have the advantage over photons in that they have no exit dose. At his institution, various plans of treatment are used and the voltage ranges from 6 mev. to 22 mev. Hot spots at adjoining fields are avoided by using polystyrene wedges and using a combination of energies of 6 mev. and 15 mev. for adjacent fields.

Dr. Melvin L. Griem of the University of Chicago related his experience with a linear accelerator ranging from 5 mev. to 55 mev. He demonstrated improvement in biologic effect on normal tissue in the region of the irradiated carcinoma of the nasal cavity, urinary bladder, larynx, parotid gland and glomus tumor.

Dr. Norah DuV. Tapley and Dr. Gilbert H. Fletcher of M. D. Anderson Hospital in Houston have treated 668 patients with malignant tumors of the head, neck and breast with high speed electrons at levels varying from 6 mev. to 18 mev. The electron beam alone was used in 284 patients, the electron beam in combination with photons in 148 patients, and an electron beam boost in 236 patients. Dr. Fletcher outlined the evolution of the use of electron beam therapy at M. D. Anderson Hospital and showed many color slides of treated patients.

During the morning hours of the second day, the ladies enjoyed a bus tour which included the Apache Trail and Roosevelt Dam area. A Home and Ranch tour was held in the afternoon which included the home of Senator Goldwater and the Brusally Arabian Horse Ranch. In the evening, a Western Style Chuck Wagon Dinner was arranged for the membership in the open desert north of the Camel Back Inn.

The third scientific session on Friday morning was held under the chairmanship of Dr. Juan A. del Regato. Subjects reported on included the treatment of primary adenocarcinoma of the cervix; carcinoma of the floor of the mouth; a direct reading intracavitary dosimeter for use in radium therapy; antibodies after local irradiation; the value of liver scans in the management of patients with malignant lymphoma; the search for hidden cancer; cobalt 60 teletherapy of early carcinomas of the vocal cords; hyperbaric radiation therapy, residual radiopaque bolus in managing intraspinal neoplasms; radiation related to epidemiology of cancer; and a short historical review of the American Radium Society by Dr. Edith H. Quimby.

The highlight of the meeting was the presentation of the Annual Janeway Lecture. Dr. Gordon P. McNeer was unable to appear personally due to sudden illness. Dr. William S. MacComb introduced Dr. McNeer *in absentia* giving a brief historical background of his medical career. Dr. Lemuel Bowden, long-time associate, then presented Dr. McNeer's prepared lecture on "The Problem of the Local Recurrence of Malignant Melanoma." Following a detailed statistical analysis, he summarized the problem by the following statements: Inadequate treatment of melanoma delays definitive management and increases the hazard of dissemination to regional lymph nodes and distant organs. Adequate treatment of melanoma is followed by local failure in 9 per cent of patients in Stage I and 21 per cent in Stage II. Increased occurrences of local failure associated with melanoma of the head and neck, fingers and soles of feet are probably due to inadequate excision. Local failure is more commonly observed after treatment of melanoma below, than above the elbows and knees. Local failure occurs all too frequently after amputation for Stage II melanoma located proximal to the knees and elbows. Local failure occurs more commonly after disseparate than in-continuity lymph node dissections in Stage II; but the difference is not remarkable. There is no basis for performing deferred as opposed to simultaneous discontinuous lymph node dissections as far as the rate of local failure is concerned. Seventy-nine per cent of local failures occur within the first 2 years of definitive surgery and among these cases the mortality is high. When local failure occurs after 2 years, the opportunity for long term survival is enhanced. It may be possible to decrease the incidence of local failure by more adequate primary surgical treatment, but the most promising preventative of local failure is early recognition of melanoma and prompt surgical resection before lymphatic metastases will have occurred.

In the evening, a reception for members and guests was held in the beautiful Terrace Garden of the Camel Back Inn, sponsored by the Radium Chemical Company of New York.

At the Annual Banquet, Dr. Justin J. Stein, master of ceremonies, introduced the foreign guests from France, England, Switzerland and Sweden. Also, 22 individuals who have been members of the Society for 25 years or more were introduced. A Scroll was presented to commemorate the late Mr. Joseph A. Kelley of the Radium Chemical Company. The Scroll was received by Mr. Charles Bergesch, son-in-law of the late Mr. Kelley. Dr. Stein, in eloquent terms and with appropriate ceremony, presented the Janeway Medal to Dr. Gordon McNeer (received by Dr. Lemuel Bowden) for his outstanding achievements in the field of cancer. Dr. Jerzy Einhorn, Deputy Director of the Radiumhemmet, said a few words regarding the close relationship between Swedish and American radiotherapists, extended warm greetings from Drs. Berven, Kottmeier, Sievert and Jacobsson who offered their congratulations to the Society on the occasion of its 50th anniversary. He also presented Dr. Stein with a book titled *The First Fifty Years of the Radiumhemmet.* The President then expressed his gratitude on behalf of the Society to Dr. R. Lee Foster and the members of his Arrangements Committee for the efforts in extending their gracious hospitality. Entertainment was provided by a group of Indian boys from St. John's Indian School who performed a series of original Indian dances. Members and guests also participated in dancing during the evening.

The fourth scientific session was called to order on Saturday morning by Dr. John L. Pool serving as chairman. The subjects presented included the synergistic effect of laser radiation and ionizing radiation of malignant tumors *in vivo* and *in vitro;* the place of radiotherapy in the treatment of malignant metastatic lymph nodes of the neck by Dr. Vera M. Dalley of the Royal Marsden Hospital, London, England; the use of radiographic methods in the classification in staging of laryngeal tumors; transvaginal cesium 137 teletherapy; and high pressure oxygenation and dose fractionation in animal tumors.

In the latter part of the morning, Dr. Franz Buschke from the University of California Medical Center talked on his 25 years of experience with supervoltage therapy in the treatment of bladder cancer. He uses a three-field fixed beam technique with a present dosage range of 5,500–6,000 rads in 5 to 7 weeks and obtains a 14 per cent 5 year survival rate with no significant morbidity. Other papers covered the treatment of pineal body tumors; management of malignant lymphomas and Hodgkin's disease limited to the head and neck; Co^{60} telecurietherapy, with and without surgery, in the definitive treatment of cancer of the testicle; the effect of external and internal irradiation on the lymphatic dynamics; preservation of the larynx in surgical treatment of cancer recurrent after radiation therapy; and the application of new radiobiology knowledge to high speed electron beam radiation therapy by Dr. Maurice Tubiana. The last paper on the program was given by Dr. Alexander Brunschwig of Memorial Hospital of New York on the surgical treatment of carcinoma of the cervix after previous failure of radiation therapy.

The Forty-eighth Annual Meeting of the American Radium Society proved successful by the large attendance, the fine quality of its scientific program and by the social and recreational attractions of the Phoenix-Scottsdale area. The members and guests of the Society owe a debt of gratitude to its President, Dr. Justin J. Stein, to Dr. R. Lee Foster and the members of his Arrangements Committee, and to the management of the Camel Back Inn, the Mountain Shadows Motel and other neighborhood motels for their efforts in providing many excellent conveniences and recreational facilities.

HARRY HAUSER, M.D.

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NEWS ITEMS

NEW OFFICERS OF THE AMERICAN RADIUM SOCIETY

At the Forty-eighth Annual Meeting of the American Radium Society held at the Camel Back Inn, Phoenix, Arizona, April 13–16, 1966, the following new officers were elected: *President*, Milton Friedman, M.D., New York, New York; *President-Elect*, John L. Pool, M.D., New York, New York; *First Vice-President*, Harry Hauser, M.D., Cleveland, Ohio; *Second Vice-President*, Daniel Catlin, M.D., New York, New York; *Secretary*, Fernando G. Bloedorn, M.D., Baltimore, Maryland; *Treasurer*, Juan A. del Regato, M.D., Penrose Cancer Hospital, 2215 North Cascade Avenue, Colorado Springs, Colorado 80907.

The Forty-ninth Annual Meeting will be held at the Royal York Hotel, Toronto, Ontario, Canada, May 29, 30 and 31, 1967.

TWELFTH INTERNATIONAL CONGRESS OF RADIOLOGY

The Twelfth International Congress of Radiology will be held October 6–11, 1969, at the Hotel New Otani, Tokyo, Japan.

Professor Dr. Tadashi Adachi, Chairman of the Department of Radiology, Tokyo Medical and Dental University School of Medicine, was nominated as the General Secretary of the Congress.

Until the Office of the Secretariat is established, any inquiry concerning the Congress should be addressed to Kempo Tsukamoto, M.D., President-Elect of the Twelfth International Congress of Radiology, Director of the National Institute of Radiological Sciences, 9-1, 4-chome, Anagawa, Chiba, Japan.

MOUNT ZION HOSPITAL AND MEDICAL CENTER

The Second Annual San Francisco Cancer Symposium on "High Energy Electrons in the Treatment of Cancer," will be held October 1 and 2, 1966, at Claire Zellerbach Saroni Memorial Tumor Institute of San Francisco Mount Zion Hospital and Medical Center.

For further information please write Jerome M. Vaeth, M.D., Director, 1600 Divisadero Street, San Francisco, California 94115.

EUROPEAN ATOMIC ENERGY COMMUNITY

The Second International Conference on *Methods of Preparing and Storing Labelled Compounds* will be held November 28–December 3, 1966, at the Palais des Congrès, Salle Benelux, Brussels, Belgium.

The final program as well as complementary general information will be circulated at the beginning of September, 1966.

Special rates for the Congress have been obtained at the Hôtel Métropole (first class) and Hôtel de la Bourse (second class). Participants are requested to make their own arrangements either through their local Cook's agent or through the Cook's office in Brussels: 17, Place de Brouckère, Bruxelles.

Correspondence and documents should be forwarded to: Euratom—Labelled Compounds, 51-53, rue Belliard, Bruxelles, Belgium.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

THE TRAIL OF THE INVISIBLE LIGHT. By E. R. N. Grigg, M.D., Attending Radiologist, Cook County Hospital, Chicago; Consulting Radiologist to the Illinois Department of Public Welfare and the hospitals in Charleston, Harvard, Paris, and Paxton, Ill. Cloth. Pp. 974, with many illustrations. Price, $36.75. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1965.

Interest in history is not a sign of mental rumination or senescence, but rather of full cognizance of our indebtedness to our predecessors, and of intellectual maturity, with an ability to bridge the past to the future, while living in and trying to understand the present.

Gifted with many and varied aptitudes, Dr. Grigg has already distinguished himself by his many contributions, and the excellence and versatility of his writing.

With boundless energy, endless capacity for work, a great penchant for accuracy and detail, and with extensive contacts in this country and abroad, Dr. Grigg has carefully selected and put together a great wealth of valuable information and perinent data between the two covers of his book. *The Trail of the Invisible Light* is of encyclopedic proportions, extending far beyond the confines and limitations of a "history book." This material, well selected, well preserved, and well presented, is invaluable to the student of radiologic history, who is now spared the trials and tribulations of searching through mountains of archives scattered all over the world.

In a very refreshing and quite personal style, the author gives a very comprehensive review of the progress attained and of the contributions—small or large—made in each country.

Beginning with Röntgen and his discovery, the history of radiology is divided into: (1) The Era of the Pioneers; (2) The Golden Age; and (3) The Atomic Phase, with the two great wars separating these three eras.

There are 8 chapters of varying lengths with an abundance of drawings, tables, charts, photographs of individuals, groups, and events.

There are innumerable biographic annotations, giving the major contributions of the pioneers in radiology and of those who made radiology what it is today. In this connection, Chapter 6 is noteworthy, as it gives the story of William Morton, the first medical electroradiologist in America, whose career took him through psychiatry into medical electricity, and then—into radiology.

Manufacturers of X-ray equipment and accessories, in this country and abroad, and their part in the steady advancement of radiology are discussed in detail. The achievements in diagnostic radiology, radiation therapy, including radium and radioactive isotopes, are likewise reviewed in full.

There is an excellent presentation of the birth and development of the National Radiologic Societies in the United States, their growing pains, their contributions to, and the significant role they have played and continue to play in American radiology particularly.

The book ends with a brief chapter on radiologic periodicals, followed by one giving an annotated bibliography on radiologic history with chronologic tables, forming a historic synopsis of radiology in the United States and Canada.

The bibliography at the end of each chapter is extensive and reflects the magnitude of the task undertaken by the author.

Dr. Grigg is a firm believer in private enterprise, individual incentive and accomplishments. On page 637, in a presentation of the accomplishments of individual radiologists, he comments on the trend to regard teamwork as a solution to most problems: "Five, seven, or eleven heads are certainly better than one, but could a committee have written Hamlet?" "Even in the peculiar world of today, one head, supported by one income, can still achieve things which to a committee might appear to be a staggering, impossible task." It is in this belief that E. R. N. Grigg, single-handed, and with one head, completed a monumental task and gave us an unparallelled legacy. To him we owe a great debt.

The Trail of the Invisible Light makes for easy, engaging, and fascinating reading. This remarkable book, covering the entire history of radiology, is unhesitatingly recommended.

William H. Shehadi, M.D.

PRINCIPLES OF BONE X-RAY DIAGNOSIS. Second Edition. By George Simon, M.D., F.R.C.P., F.F.R., Assistant Director, X-Ray Department, Brompton Hospital, London; Radiologist, St. Bartholomew's Hospital, London; Demonstrator, Radiological Anatomy, St. Bartholomew's Hospital, London; Curator of the Radiological Museum and Teacher of Radiology, The Institute of Diseases of the Chest, University of London,

London. Cloth. Pp. 193 with many illustrations. Price, $13.25. Butterworth Inc., 7300 Pearl Street, Washington 14, D. C., 1965.

This Second Edition of *Principles of Bone X-Ray Diagnosis* expands the contents of the first edition which was published in 1960. The material is grouped according to the type of x-ray findings rather than classified according to clinical diseases. A method of study of roentgenograms of the skeletal system resulting from considerations of the balance between absorption, sclerosis and periosteal new bone is utilized. Dr. Simon indicates that a study of this balance, together with the size, shape and position of a lesion, will aid the student in reducing the list of possibilities for diagnosis to a less formidable number. In addition, it will aid in indicating the need for biochemical, endocrine, histologic, hematologic and bacterial investigations which may not otherwise have been performed and which could lead to the correct diagnosis, or even result in the immediate diagnosis, before it might be established clinically.

The new edition contains 53 additional illustrations, many of which are of conditions not included in the first edition; others have been substituted to make it easier for the reader to remember and understand the principles described in the text. The objective of selecting examples which illustrate the principles of bone x-ray diagnosis, rather than completely cataloging all the conditions, is well achieved.

The size of the book precludes the possibility of including roentgenographic reproductions to illustrate every condition described, but mention is made of rare conditions as well as the general common conditions.

The book has been aimed essentially at the trainee radiologist. However, the material is such that, as a quick reference, it is a useful addition to any radiologic library. The contents are skillfully organized in 12 chapters and the illustrations are excellent. Included in the contents are complementary chapters on alterations of joint space (arthritis) and associated bone changes, with some hints on x-ray technique of bone in unusual sites and calcifications in or near a joint. Chapter 7 on "A Translucent Area in a Bone" is especially well written.

The appendage contains additional tables of classifications of bone tumors, normal blood chemistry, hematology and urinary findings, as well as handy tables for milli-equivalent conversions.

This excellent book, with many superb illustrations, can be read with profit, not only by the student, but also by the practicing radiologist.

E. Nicolas Sargent, M.D.

BOOKS RECEIVED

Charcot Joints. By Sidney N. Eichenholtz, M.A., M.D., F.A.C.S., Assistant Clinical Professor of Orthopaedic Surgery, Cornell University School of Medicine; Attending Orthopaedic Surgeon, Bronx Veterans Administration Hospital; Associate Attending Orthopaedic Surgeon, Hospital for Special Surgery; Assistant Attending Orthopaedic Surgeon, New York Hospital; Director of Orthopaedic Surgery, Yonkers Professional Hospital, Yonkers, N. Y. Cloth. Pp. 227, with many illustrations. Price, $15.75. Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Ill., 1966.

Radiological Anatomy. By D. Nagy, M.D., translated by Dr. A. Deák. Edited translation of Röntgenanatómia published in Hungary in 1959. Cloth. Pp. 516, with many illustrations. Price, $17.50. Pergamon Press, Inc., 44-01, 21st Street, Long Island City, N. Y., 1966.

Physics of Industrial Radiology. Edited by R. Halmshaw, B.Sc., A.R.C.S., A.Inst.P., Royal Armament Research and Development Establishment, Fort Halstead, Sevenoaks, Kent, England. Cloth. Pp. 491, with 191 illustrations. American Elsevier Publishing Company, Inc., 52 Vanderbilt Avenue, New York, N. Y., 1966.

The Use of Diagnostic Ultrasound in Brain Disorders. By Charles C. Grossman, M.D., Director, Laboratory of Electroencephalography and Diagnostic Ultrasound, Presbyterian-University Hospital; Senior Staff Physician, Western Pennsylvania Hospital; Associate Neurologist, Montefiore Hospital; Consultant, Veterans Administration, Magee, St. Margaret and Pittsburgh Hospitals, Pittsburgh, Pa. Cloth. Pp. 155, with many illustrations. Price, $9.00. Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Ill., 1966.

Radioisotope Sample Measurement Techniques in Medicine and Biology. Proceedings of the Symposium on Radioisotope Sample Measurement Techniques in Medicine and Biology held by the International Atomic Energy Agency, Vienna, May 24–28, 1965. Cloth. Pp. 724, with some figures. Price, $15.00. International Publications, Inc., 319 East 34th Street, New York, N. Y., 1965.

Pneumoencephalography and Cerebral Angiography. By Bernard S. Epstein, M.D., Clinical Professor of Radiology, The Albert Einstein College of Medicine, Yeshiva University, New York City; Director of Radiology, The Long Island Jewish Hospital, New Hyde Park, N. Y.; Radiologist-in-Chief, The Long Island Jewish Hospital-Queens Hospital Center Affiliation, Queens, N. Y. Cloth. Pp. 349, with many illustrations. Year Book Medical Publishers, 35 East Wacker Drive, Chicago, Ill., 1966.

Radiographic Atlas of the Human Skull: Normal Variants and Pseudo-Lesions. By Gerhart S. Schwarz, M.D., Chief of Radiologic Services, New York Medical College of Chronic Disease Center, Bird S. Coler Hospital, and Professor of Radiology, New York Medical College; Attending Radiologist, Flower and Fifth Avenue Hospitals; Visiting Radiologist, Metropolitan Hospital; and Charles R. Golthamer, M.D., Radiologist, Van Nuys, California. Cloth. Pp. 60, plus 12 large plates. Price, $42.50. Hafner Publishing Company, Inc., 31 East 10th Street, New York, N. Y., 1965.

Splenoportography: Diagnostic Phlebography of the Portal Venous System. By Lucien Leger, M.D., Professor of Clinical Surgery, Medicine Faculty of Paris; Chief Surgeon of Cochin Hospital, Paris; Member of French Academy of Surgery. American Lecture Series, No. 634. Edited by I. Newton Kugelmass, M.D., Ph.D., Sc.D., Consultant, Departments of Health and Hospitals, New York, N. Y. Cloth. Pp. 121, with many illustrations. Price, $8.50. Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Ill., 1965.

Pathology for Radiographers. By Dr. G. Ch. F. Brinkbok, Radioloog aan het, Nederlands Hervormd Diaconessenhuis te Arnhem. Translated by May Hollander. Cloth. Pp. 231, with some illustrations. Price, $10.75. Butterworth Inc., 7300 Pearl Street, Washington, D. C., 1966.

Die angeborenen Fehlbildungen der Hand und ihre operative Behandlung. By Prof. Dr. Alfred N. Witt, Direktor der Orthopädischen Klinik und Poliklinik der Freien Universität Berlin im Oskar-Helene-Heim; Priv.-Doz. Dr. Horst Cotta, Oberarzt der Orthopädischen Klinik und Poliklinik der Freien Universität Berlin im Oskar-Helene-Heim; and Dr. Michael Jäger, Wiss. Assistent der Orthopädischen Klinik und Poliklinik der Freien Universität Berlin im Oskar-Helene-Heim. Cloth. Pp. 174, with 92 illustrations. Price, Ganzleinen DM 57.-. Georg Thieme Verlag, Stuttgart. In the U.S.A. and Canada, Intercontinental Medical Book Corporation, New York 16, N. Y., 1966.

Developmental and Metabolic Control Mechanisms and Neoplasia. Collection of papers presented at the Nineteenth Annual Symposium on Fundamental Cancer Research, 1965. Published for The University of Texas M. D. Anderson Hospital and Tumor Institute. Cloth. Pp. 514, with many illustrations. Price, $16.00. The Williams and Wilkins Company, Baltimore, Md., 1966.

Introduction à la Scintigraphie Clinique: Atlas. By B. Delaloye, with the collaboration of P. Magnenat, B. Scazziga, and G. Gauthier. Paper. Pp. 262, with 192 illustrations. Price, 84 F. Masson & Cie, Éditeurs, 120 Boulevard Saint-Germain, Paris, 1966.

Clinical Foot Roentgenology: an Illustrated Handbook. By Felton O. Gamble, D.S.C., F.A.C.F.R., Ed.D.(Hon.), Professor Emeritus of Roentgenology, Temple University, School of Chiropody; Past President and Fellow, American College of Foot Roentgenology; Past President, American Podiatry Association; Formerly, Chief of Foot Clinic, Metabolic Department, Presbyterian Hospital, Philadelphia; and Irving Yale, D.S.C., F.A.C.F.R., Ed.D.(Hon.), Vice-president, American Podiatry Association; Past President and Fellow, American College of Foot Roentgenology; Formerly, Lecturer in Roentgenology, Long Island University, College of Podiatry; Formerly, Chief of Podiatry Service, Orthopedic Section, Department of Surgery, Yale-New Haven Community Hospital; Fellow, American Public Health Association. Cloth. Pp. 295, with 412 illustrations. Price, $15.50. Williams and Wilkins Company, Baltimore, Md., 1966.

Manual de Telecobaltoterapia. By Dr. Raul Vera, A.B.R., A.R.S., A.C.R., S.V.O., S.V.C., S.V.U., Profesor Titular, Cátedra de Clínica Electro-Radiológica, Sección de Radioterapia y Radioisótopos, Facultad de Medicina, Universidad Central de Venezuela, Caracas; Jefe del Departamento de Radiaciones, Instituto de Oncología "Luis Razetti," Caracas; and Dr. Modesto Rivero Gonzalez, S.V.R., S.V.O., S.V.C., S.V.M.I., S.V.U., Profesor Agregado, Cátedra de Clínica Electro-Radiológica, Sección de Radioterapia y Radioisótopos, Facultad de Medicina, Universidad Central de Venezuela, Caracas; Médico-Adjunto del Departamento de Radiaciones, Instituto de Oncología "Luis Razetti," Caracas. Cloth. Pp. 141, with 68 illustrations. Libreria University, Apartado 10786, Caracas, Venezuela, 1966.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. C. Allen Good, Mayo Clinic, Rochester, Minn. Annual meeting: San Francisco Hilton Hotel, San Francisco, Calif., Sept. 27–30, 1966.

American Radium Society
Secretary, Dr. Fernando G. Bloedorn, Division of Radiotherapy, University of Maryland Hospital, Baltimore, Md. 21201. Annual Meeting: Royal York Hotel, Toronto, Ont., Canada, May 29–31, 1967.

Radiological Society of North America
Secretary-Treasurer, Dr. Maurice Doyle Frazer, 1744 South Fifty-eighth St., Lincoln, Neb. Annual meeting: Palmer House, Chicago, Ill., Nov. 27–Dec. 2, 1966.

American College of Radiology
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Ill. Annual meeting: Beverly Hilton Hotel, Los Angeles, Calif., Jan. 21–Feb. 4, 1967.

Section on Radiology, American Medical Association
Secretary, Dr. Clyde A. Stevenson, Sacred Heart Hospital, West 101 Eighth Ave., Spokane 4, Wash. Annual meeting: Chicago, Ill., June 26–30, 1966.

American Board of Radiology
Secretary, Dr. H. Dabney Kerr. Correspondence should be directed to Kahler Hotel Building, Rochester, Minn.

The Spring 1966 examination will be held at the Terrace Hilton Hotel, Cincinnati, Ohio, June 6–10, inclusive. The deadline for filing applications for this examination was December 31, 1965.

The Fall 1966 examination will be held at the Washington Hilton Hotel, Washington, D.C., December 5–9, inclusive. The deadline for filing applications is June 30, 1966.

American Association of Physicists in Medicine
Secretary, Leonard Stanton, Hahnemann Medical College, 230 N. Broad St., Philadelphia, Pa. 19102. Annual meeting to be announced.

American Club of Therapeutic Radiologists
Secretary, Dr. J. A. del Regato, Penrose Cancer Hospital, Colorado Springs, Colo.

Twelfth International Congress of Radiology
President, Dr. Kempo Tsukamoto, 9-1, 4-chome, Angawa, Chiba, Japan. Meeting: Hotel New Otani, Tokyo, Japan, Oct. 6–11, 1969.

Ninth Inter-American Congress of Radiology
Counselor for the United States, Dr. Philip J. Hodes, Jefferson Medical College Hospital, 11th and Walnut Streets, Philadelphia 7, Pennsylvania.
President, Dr. Leandro Zubiaurre, Montevideo, Uruguay. Meeting: Montevideo, Uruguay, 1967.

Inter-American College of Radiology
President, Dr. Oscar Soto, H. Urteaga 480, Lima, Perú.

Alabama Radiological Society
Secretary, Dr. Walter Brower, Birmingham, Ala. Meets time and place of Alabama State Medical Association.

American Nuclear Society
Treasurer, Raymond Maxson, 86 E. Randolph St. Chicago, Ill. Annual meeting to be announced.

American Society for Diagnostic Ultrasound
Secretary, Dr. Charles C. Grossman, 552 N. Neville St., Pittsburgh, Pa. 15213.

Arizona Radiological Society
Secretary-Treasurer, Dr. George Gentner, 3435 W. Durango, Phoenix, Ariz. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

Arkansas Chapter of American College of Radiology
Secretary-Treasurer, Dr. James R. Morrison, 550 S. University, Little Rock, Ark. 72205.

Arkansas Radiological Society
Secretary, Dr. Charles W. Anderson, 1108½ Poplar, Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

Association of University Radiologists
Secretary-Treasurer, Dr. Morton M. Kligerman, Dept. of Radiology, Yale University Medical School, New Haven, Conn. Annual meeting—to be announced.

Atlanta Radiological Society
Secretary, Dr. Donald R. Rooney, Burnt Hickory Road, Marietta, Ga. Meets monthly except during three summer months, on third Tuesday, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

Bavarian-American Radiologic Society
Secretary, Dr. Roy R. Deffebach, Major, MC, Radiology Service, 5th General Hospital, APO 154, New York, N. Y. Meets quarterly.

Blockley Radiological Society
Secretary-Treasurer, Dr. R. John Gould, 441 Lombardy Rd., Drexel Hill, Pa. 19026.

Bluegrass Radiological Society
Secretary-Treasurer, Dr. Arthur Lieber, University of Kentucky, University Hospital, Lexington, Kentucky. Meets quarterly.

Brooklyn Radiological Society
Secretary, Dr. Edward Feely, St. Johns Episcopal Hospital, 480 Herkimer St., Brooklyn, N. Y. Meets first Thursday of each month, October through June.

Buffalo Radiological Society
Secretary, Dr. Richard Munschauer, 130 Hodge Ave., Buffalo, N.Y. 14222. Meets second Monday evening each month, October to May inclusive.

California Radiological Society
Secretary, Dr. L. Henry Garland, Suite 1739, 450 Sutter St., San Francisco, Calif. Meets annually during meeting of California Medical Association.

Catawba Valley Radiological Society
Secretary, Dr. Emmett R. White, P. O. Box 303, Rutherford College, N. C. Meets every Tuesday, Dept. of Radiology, Valdese General Hosp., Valdese, N. C. at 12:00 P.M.

Central New York Radiological Society
Secretary-Treasurer, Dr. Robert A. Bornhurst, State Univ. Hospital, 750 E. Adams St., Syracuse, N.Y. 13210. Meets first Monday each month, October through May.

Central Ohio Radiological Society
Secretary-Treasurer, Dr. Atis K. Freimanis, Ohio State Univ. Hospitals, 410 W. 10th Ave., Columbus, Ohio 43210. Meets second Thursday in October, November, January, and March 15 and May 19 at Fort Hayes Hotel, Columbus, Ohio.

Central Society of Nuclear Medicine
Secretary, Dr. Robert S. Landauer, Radiation Center Bldg., 1903 West Harrison St., Chicago 12, Ill

Chicago Roentgen Society
Secretary-Treasurer, Dr. Fredric D. Lake, 2548 N. Lakeview Ave., Chicago, Ill. 60614. Meets second Thursday of each month, October to April except December at the Pick-Congress Hotel at 8:00 P.M.

Cleveland Radiological Society
Secretary-Treasurer, Dr. James Christie, 10515 Carnegie Avenue, Cleveland, Ohio. Meetings at 7:00 P.M. on fourth

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*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Monday of October, November, January, February, March and April.

Colorado Radiological Society
Secretary, Dr. George F. Wertz, 1801 High St., Denver, Colo. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society
Secretary, Dr. William W. Walthall, Jr., 130 Maple St., Springfield, Mass. Meets in April and October.

Dallas-Fort Worth Radiological Society
Secretary-Treasurer, John S. Alexander, 1217 W. Cannon, Ft. Worth, Tex. Meets monthly, third Monday, at Southwest International Airport at 6:30 P.M.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. Robert L. Willis, Harper Hospital, Detroit 1, Mich. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

East Bay Radiological Society
Secretary, Dr. Tom H. Piatt, 12 Camino Encinas, Orinda, Calif. 94563. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society
Secretary, Dr. C. H. Kimball, 2200 Harris Circle, Cleveland, Tenn. Meets in January and September.

Eastern Radiological Society
Secretary, Dr. James F. Martin, North Carolina Baptist Hospital, Winston-Salem, N. C.

Florida Radiological Society
Secretary, Dr. John C. Jowett, Orlando, Fla. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. Garth R. Drewry, Tampa General Hospital, Tampa 6, Fla. Meets in January, April, July and October.

Georgia Radiological Society
Secretary, Dr. I. R. Berger, 1010 Prince Ave., Athens, Ga. Meets in spring and fall with Annual State Society Meeting.

Greater Miami Radiological Society
Secretary-Treasurer, Dr. Arthur R. Miller, North Miami General Hospital, 1701 N.E. 127th St., North Miami, Fla. Meets monthly, third Wednesday at 8:00 P.M. at Jackson Memorial Hospital, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary-Treasurer, Dr. John W. Fries, 3520 Chippewa St., St. Louis, Mo. 63118.

Houston Radiological Society
Secretary, Dr. William A. Vint, 1004 Seymour, Pasadena, Tex. Meets fourth Monday of each month, except June, July, August and December, at the Doctors' Club, 8:00 P.M., Houston, Tex.

Idaho State Radiological Society
Secretary, Dr. George H. Harris, Bannock Memorial Hospital, Pocatello, Idaho. Meets in the spring and fall.

Illinois Radiological Society
Secretary, Dr. George A. Miller, Carle Hospital Clinic, Urbana, Ill. Meets in the spring and fall.

Indiana Roentgen Society, Inc.
Secretary, Dr. Richard A. Silver, 1815 N. Capitol Avenue, Indianapolis, Ind. Meets first Sunday in May and during fall meeting of Indiana State Medical Association.

Iowa Radiological Society
Secretary, Dr. L. L. Maher, 1419 Woodland Ave., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society
Secretary-Treasurer, Dr. Robert C. Lawson, 310 Medical Arts Bldg., 10th and Horne, Topeka, Kan. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter, American College of Radiology
Secretary-Treasurer, Dr. Robert H. Greenlaw, Dept. of Radiology, Univ. of Kentucky Med. Ctr., Lexington, Ky. Meets semiannually.

Kentucky Radiological Society
Secretary-Treasurer, Dr. Joan R. Hale, 402 Heyburn Building, Louisville, Ky. Meets monthly on second Friday at Sheraton Hotel, Louisville, Ky.

Kings County Radiological Society
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society
Secretary, Dr. David Faegenburg, Nassau Academy of Medicine, 1200 Stewart Ave., Garden City, L. I., N. Y. 11533. Meets second Tuesday of the month in February April, June, October and December.

Los Angeles Radiological Society
Secretary, Dr. Joseph A. Parks, 15107 Vanowen St., Van Nuys, Calif. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif.

Louisiana-Texas Gulf Coast Radiological Society
Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Texas 77701.

Maine Radiological Society
Secretary, Dr. J. T. Chen, 7 Cherry Hill Terrace, Waterville, Me. Meets in June, September, December and April

Maryland Radiological Society
Secretary, Dr. Henry Startzman, Medical Arts Building, Baltimore, Md.

Memphis Roentgen Society
Secretary-Treasurer, Dr. Vernon I. Smith, Jr., Suite 203, 1085 Madison Ave., Memphis, Tenn. 38104. Meets first Monday of each month at John Gaston Hospital.

Miami Valley Radiological Society
Secretary, Dr. Darwood B. Hance, Reid Memorial Hospital, Richmond, Indiana. Meets third Tuesday of fall and winter months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

Mid-Hudson Radiological Society
Secretary-Treasurer, Dr. Alexander W. Friedman, Mid-Hudson Medical Group, Fishkill, N. Y. Meets 7:00 P.M., first Wednesday of each month, September to May.

Milwaukee Roentgen Ray Society
Secretary-Treasurer, Dr. Donald P. Babbitt, 1700 W. Wisconsin Ave., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society
Secretary-Treasurer, Dr. Edward A. Peterson, 572 Lowry Medical Arts Bldg., St. Paul, Minn. Meets twice annually, fall and winter.

Mississippi Radiological Society
Secretary-Treasurer, Dr. Dan T. Keel, Jr., 504 Chippewa St., Brookhaven, Miss. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 P.M.

Missouri Radiological Society
Secretary-Treasurer, Dr. M. Shoss, Cape Girardeau, Mo.

Montana Radiological Society
Secretary, Dr. Clark Grimm, Great Falls, Montana. Meets at least once a year.

Nebraska State Radiological Society
Secretary, Dr. Richard Bunting, The Radiologic Center, Nebraska Methodist Hospital, Omaha 31, Neb. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

Nevada Radiological Society
Secretary, Dr. William G. Arbonies, Department of Radiology, St. Mary's Hospital, Reno, Nev.

New England Roentgen Ray Society
Secretary, Dr. Jack R. Dreyfuss, Zero Emerson Place, Boston, Mass. 02114. Meets third Friday of each month, October through April, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 4:30 P.M.

New Hampshire Roentgen Ray Society
Secretary, Dr. Paul Y. Hasserjian, 1470 Elm St., Manchester, N. H. Meets four to six times yearly.

New Mexico Association of Radiologists
Secretary-Treasurer, Dr. Justin J. Wolfson, Department of Radiology, Bernalillo County-Indian Hospital, Albuquerque, New Mexico.

New Mexico Society of Radiologists
Secretary, Dr. William G. McPheron, Hobbs, New Mexico. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New York Roentgen Society
Secretary, Dr. Milton Elkin, Albert Einstein College of Medicine, Bronx, N.Y. 10461. Meets monthly on third Monday at the New York Academy of Medicine at 4:30 P.M. Annual meeting to be announced.

North Carolina Radiological Society
Secretary, Dr. E. H. Schultz, North Carolina Memorial Hospital, Chapel Hill, N. C. Meets in the spring and fall each year.

North Dakota Radiological Society
Secretary, Dr. Robert J. Olson, 1240 8th Ave., Williston, N. D. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. Charles H. Newell, 800 Miami Road, Jacksonville 7, Fla. Meets quarterly in March, June, September and December.

Northeastern New York Radiological Society
Secretary, Dr. Anthony J. Tabacco, 621 Central Ave., Albany 6, N. Y. Meets in Albany area on second Wednesday of October, November, March and April.

Northern California Radiological Society
Secretary-Treasurer, Dr. John Turner, 1215-28th St., Sacramento, Calif. Meets fourth Monday of Sept., Nov., Jan., March and May at the Sutter Club in Sacramento.

Northwestern Ohio Radiological Society
Secretary, Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

Ohio State Radiological Society
Secretary, Dr. Mortimer Lubert, Cleveland, Ohio. Annual meeting to be announced.

Oklahoma State Radiological Society
Secretary, Dr. Robert Sukman, 1200 N. Walker, Oklahoma City, Okla. Meets in January, May and October.

Orange County Radiological Society
Secretary, Dr. George W. Logan, 301 Newport Blvd., Newport Beach, Calif. Meets fourth Tuesday of every month at Orange County Medical Association Building.

Oregon Radiological Society
Secretary-Treasurer, Dr. Robert S. Miller, 13753 S.W. Farmington Rd., Beaverton, Ore. 97005. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary-Treasurer, Dr. Willis Taylor, 1118 9th Ave., Seattle, Washington. Annual meeting to be announced.

Pennsylvania Radiological Society
Secretary, Dr. T. Frederick Weiland, Jr., 619 Ridgeway Ave., Grove City, Pa. Annual meeting to be announced.

Philadelphia Roentgen Ray Society
Secretary, Dr. C. Jules Rominger, Misericordia Hospital, 54th St. and Cedar Ave., Philadelphia, Pa. 19143. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Robert N. Berk, 3305 Fifth Ave., Pittsburgh 13, Pa. Meets second Wednesday of month, October through June at Park Schenely Restaurant.

Radiological Society of Connecticut, Inc.
Secretary-Treasurer, Dr. Orlando F. Gabriele, 1450 Chapel St., New Haven 11, Conn. Meetings are held quarterly.

Radiological Society of Greater Cincinnati
Secretary, Dr. Harold N. Margolin, 6159 Tulane Road, Cincinnati, Ohio. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of Greater Kansas City
Secretary, Dr. J. Stewart Whitmore, 1010 Rialto Bldg., Kansas City, Mo. Meets last Friday of each month.

Radiological Society of Hawaii
Secretary-Treasurer, Dr. Robert W. Edland, P.O. Box 282, USA Tripler General Hospital, Honolulu, Hawaii. Meets third Monday of each month at 7:30 P.M.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana
Secretary, Dr. Lester W. Eavenson, 2700 Napoleon Ave., New Orleans 15, La. Meets semiannually, during Louisiana State Medical Society meeting and 6 months later.

Radiological Society of New Jersey
Secretary, Dr. E. Arthur Kratzman, 912 Prospect Ave., Plainfield, N. J. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of Rhode Island
Secretary-Treasurer, Dr. John M. Vesey, 1196 Elmwood Ave., Cranston, R. I.

Radiological Society of South Dakota
Secretary-Treasurer, Dr. Donald J. Peik, 303 S. Minnesota Ave., Sioux Falls, S. D.

Radiological Society of Southern California
Secretary-Treasurer, Dr. Gerald M. McDonnel, U.C.L.A. Medical Center for Health Sciences, Los Angeles, Calif. 90024. Meets three times a year, usually October, February, and May.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester 18, N. Y.

Redwood Empire Radiological Society
Secretary, Dr. Lee F. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. W. F. Hamilton, Jr., University Hospital, Augusta, Ga. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Irving B. Joffe, Rochester General Hospital, 1425 Portland Ave., Rochester 21, N. Y. Meets at 8:15 P.M. on the last Monday of each month, September through May, at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary-Treasurer, Dr. Robert W. Lackey, 4200 E. Ninth Ave., Denver, Colo. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 18–20, 1966.

San Antonio-Military Radiological Society
Secretary, Dr. Hugho F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Tex. Meets third Wednesday of each month in Fort Sam Houston Officer's Club at 6:30 P.M.

San Diego Radiological Society
President-Secretary, Charles P. Hyslop, 7901 Frost St., San Diego 22, Calif. Meets first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary, Dr. H. Joachim Burhenne, Children's Hospital and Adult Medical Center, 3700 California St., San Francisco, Calif. 94119. Meets quarterly at the San Francisco Medical Society, 250 Masonic Ave., San Francisco, Calif. 94118.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. George T. Hennessey, Washington, D. C. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Southern Medical Association
Secretary, Dr. Andrew F. Giesen, Jr., White-Wilson Clinic, Fort Walton Beach, Fla. Annual meeting: Washington, D. C., Nov. 14–17, 1966.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary, Dr. John L. Gwinn, Children's Hospital, 4614 Sunset Blvd., Los Angeles 27, Calif. Annual meeting: San Francisco Hilton Hotel, San Francisco, Calif., Sept. 26, 1966.

Society of Nuclear Medicine
Secretary, Mr. C. Craig Harris, Oak Ridge National Laboratories, Oak Ridge, Tenn. *Administrator*, Mr. Samuel N. Turiel, 430 N. Michigan Ave., Chicago 11, Ill. Annual meeting to be announced.

South Bay Radiological Society
Secretary, Northern Section: Dr. John H. Callaghan, 2900 Whipple Ave., Redwood City, Calif.; Southern Section: Dr. Carleton J. Wright, 2015 Clarman Way, San Jose, Calif. Meets second Wednesday of each month.

South Carolina Radiological Society
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Dakota Radiological Society
Secretary, Dr. Donald J. Peik, 1417 S. Minnesota Ave., Sioux Falls, S. Dak. Meets in spring with State Medical Society and in fall.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 4097, Mobile, Ala. Annual meeting to be announced.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society
Secretary-Treasurer, Dr. E. K. Carter, Holston Valley Community Hosp., Kingsport, Tenn. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas Radiological Society
Secretary, Dr. Herman C. Sehested, 815 Medical Arts Bldg., Fort Worth 2, Tex. Annual meeting to be announced.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital, Ann Arbor, Mich.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society
Secretary, Dr. Carlisle C. Smith, Salt Lake General Hospital, 2033 S. State St., Salt Lake City, Utah. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society
Secretary, Dr. John R. Williams, 160 Allen St., Rutland, Vt.

Virginia Radiological Society
Secretary, Dr. John M. Ratliff, Mary Immaculate Hospital, Newport News, Va.

Washington State Radiological Society
Secretary, Dr. Owen Marten, 930 Terry Avenue, Seattle, Wash. Meets quarterly.

West Virginia Radiological Society
Secretary, Dr. Karl J. Myers, The Myers Clinic-Broaddus Hospital, Philippi, W. Va. Meets concurrently with Annual Meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. Arnold Gerson, Medical Arts Bldg., Mt. Vernon, N. Y. Meets on third Tuesday of January and October and on two other dates.

Wisconsin Radiological Society
Secretary-Treasurer, Harold F. Ibach, 2400 W. Villard Ave., Milwaukee, Wis. Meets twice a year, May and September.

Wyoming Radiological Society
Secretary, Dr. Ronald R. Lund, 240 W. 9th St., Casper, Wyo. Meets in fall with State Medical Society and in spring on call of President.

CUBA, MEXICO, PUERTO RICO AND CENTRAL AMERICA

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá.
Secretary-General, Dr. Roberto Calderón, Calle Central Oeste No. 218, Managua, Nicaragua, Central America. Meets annually in a rotating manner in the six countries.

Sociedad de Radiología de El Salvador
Secretary, Dr. Rafael Vaga Gómez.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad de Radiología y Fisioterapía Cubana
Secretary, Dr. Miguel A. García Plasencia, Hospital Curie, 29 y F, Vedado, Habana, Cuba. Meets monthly at Curie Hospital.

Sociedad Costarricense de Radiologia
Secretary, Dr. James Fernández Carballo, Apartado VIII, San José, Costa Rica.

Sociedad Mexicana de Radiología, A.C.
Calle del Oro No. 15, México 7, D. F.
Secretary-General, Dr. E. Alvarez Hernández.
Meets first Monday of each month.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Jorge Carreras Girard, Suite 504, Professional Bldg., Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

BRITISH COMMONWEALTH OF NATIONS

Association of Radiologists of the Province of Quebec
Secretary, Dr. R. Robillard, Notre-Dame Hospital, 1560 Sherbrooke St., East, Montreal, Que., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretary, Dr. G. H. du Boulay, 32 Welbeck St., London, W. 1, England. Meets monthly from October until May. Annual meeting to be announced.

Canadian Association of Physicists, Division of Medical and Biological Physics.
Honorary Secretary-Treasurer, Paul M. Pfalzner, Dept. of Therapeutic Radiology, University of Western Ontario, London, Ont., Canada. Annual meeting to be announced.

Edmonton and District Radiological Society
Secretary, Dr. B. V. Evans, 105 Northgate Bldg., Alberta, Canada. Meets second Tuesday of each month in various Edmonton Hospitals.

Faculty of Radiologists
Honorary Secretary, Dr. J. N. Pattinson, 47 Lincoln's Inn Fields, London, W.C.2, England. Annual meeting to be announced.

Faculty of Radiologists, Royal College of Surgeons in Ireland

Registrar, Dr. H. O'Flanagan, F.R.C.P.I., D.P.H., 123 St. Stephens Green, Dublin 2, Ireland.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 p.m. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1, England.

Canadian Association of Radiologists
Honorary Secretary-Treasurer, Dr. Maurice Dufresne, *Associate Honorary Secretary-Treasurer*, Dr. F. Robert MacDonald, 1555 Summerhill Ave., Montreal 25, Que., Canada. Annual meeting to be announced.

Montreal Radiological Study Club
Secretary, Dr. Leonard Rosenthall, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française de Radiologie
Secretary General, Dr. Jacques Lespérance, 1656 Sherbrooke East, Montreal, Que., Canada. Meets every third Tuesday from October to April. Annual meeting to be announced.

Toronto Radiological Society
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

College of Radiologists of Australasia
Honorary Secretary, Dr. E. A. Booth, c/o British Medical Agency, 135 Macquarie St., Sydney, N.S.W., Australia.

South America

Asociación Argentina de Radiología
Secretary, Dr. Lidio G. Mosca, Avda. Gral. Paz 151, Córdoba, Argentina. Meetings held monthly.

Ateneo de Radiologia
Secretary, Dr. Victor A. Añaños, Instituto de Radiologia, Santa Fe 3100, Rosario, Argentina. Meets monthly on second and fourth Fridays at 7:00 p.m. in the Hospital Nacional de Centenario, Santa Fe 1300, Rosario.

Colégio Brasileiro de Radiologia
Secretary-General, Dr. Tede Eston de Eston, Caixa Postal 5984, São Paulo, Brazil.

Sociedad Argentina de Radiologia
Secretary, Dr. Edwarde A. Navarrine, Santa Fe 1171, Buenos Aires. Meetings are held monthly.

Sociedad Bolivana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1596, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Nicola Caminha, Av. Mem. de Sa, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 p.m. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. J. P. Velasco, Avenida Santa María 0410, Santiago, Chile. Meets fourth Friday of each month.

Sociedad Colombiana de Radiologia
Secretary, Dr. Armando Uribe, Hospital Miltar Central, Apartado aéreo No. 5804, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología y Fisioterapía
Secretary, Dr. Luis Blum, P.O. Box 3712, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Miguel González Addone, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary, Dr. Vicente Ubillus, Apartado 2306, Lima, Peru. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta 218, Lima.

Sociedad de Radiologica del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Barranquilla, Colombia. Society meets monthly at the Instituto de Radiologia.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Ernesto H. Cibils, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiologia de Pernambuco
Secretary, Dr. Manoel Medeiros, Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenología y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz 151, Córdoba, Argentina.

Sociedad Venezolana de Radiología
Secretary-General, Dr. Luis F. Muro, Apartado No. 9362 Candelaria, Caracas, Venezuela. Meets monthly third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
President, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik. Annual meeting to be announced.

Société Belge de Radiologie
General Secretary, Prof. Simon Masy, Louvain, Belgium. Meets in February, March, May, June, September, October, November and December.

Société Européenne de Radiologie Pédiatrique
Permanent Secretary, Dr. Jaques Sauvegrain, Hôpital des Enfants-Malades, 149, rue de Sèvres, Paris 15e, France. *General Secretary*, Dr. Ole Eklöf, P.O. Box, Stockholm 60, Sweden. Annual meeting to be announced.

Société Française d'Électroradiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord. Central Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru, Paris 8e, France.

Československá Společnost pro Roentgenologii a Radiologii
Secretary, Dr. Robert Poch, Praha 12, Srobárova 50, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Röntgengesellschaft
Secretary, Professor Dr. med. H. Lossen, Universitäts-Röntgeninstitut, Langenbeckstr. 1, Mainz, Germany.

Società Italiana di Radiologia Medica e di Medicina Nucleare
Secretary, Dr. Ettore Conte, Ospedale Mauriziano, Torino, Italy. Meets annually.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. H. F. O. Stricker, Schalklaar, Netherlands.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Española de Radiología y Electrología Médicas y Medicina Nuclear
Secretary, Dr. D. Aureo Gutierrez Churruca, Esparteros, No. 9, Madrid, Spain. Meets monthly in Madrid.

Schweizerische Gesellschaft für Radiologie und Nuklearmedizin (Société Suisse de Radiologie et de Médecine Nucléaire)
Secretary, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

Asia

Indian Radiological Association
Secretary, Dr. R. F. Sethna, Navsari Building, Hornby Road, Bombay 1, India.

Indonesian Radiological Society
Secretary, Professor Sjahriar Rasad, Taman Tjut Mutiah 1, Djakarta, Indonesia.

ABSTRACTS OF RADIOLOGICAL LITERATURE

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ROENTGEN DIAGNOSIS

HEAD

VIGNAUD, J., DANIC, J., LICHTENBERG, R., and SUDAKA, J. Nouvelles recherches en radio-otologie. I. Radiographie de l'étrier. II. Aspect tomographique de la fenêtre ovale dans l'otospongiose. (New research in radio-otology. I. Radiography of the stapes. II. Tomographic aspect of the oval window in otospongiosis.) *J. de radiol., d'électrol. et de méd. nucléaire*, March–April, *1965*, 46, 129–134. (From: Service de Radiologie et Service d'O.-R.-L. de la Fondation a.-de-Rothschild, 29, rue Manin, Paris, 19e, France.)

Following the International Symposium held in Bordeaux in April 1963 on the radiology in ORL, a program was devised under Professor Fishgold for radiologic investigation of the stapes.

In the present article, the authors present their first results. Tomography with the polytome, using the hypocycloidal movement and sections at each millimeter, proves to be the best way to demonstrate the stapes and to study the oval window (focus 0.6 mm., absence of grid, strong collimation). The most suitable incidence is Guillen's transorbital position (20° angle with the sagittal plane). In this position, the inner wall of the tympanic cavity, the oval fossa and the oval window are visualized perpendicular to the film. The stapes forms a "V" shape with the descending portion of the incus.

The authors were able to demonstrate the stapes in 69 per cent of cases in 56 consecutive examinations. When the ½ mm. cuts become available, it is probable that it will be seen regularly.

Such precision radiography permits the study of the oval fossa, the second portion of the fallopian canal, and the recognition of the changes caused by otospongiosis, as well as the degree of sclerosis of the margins of the fenestra, the narrowing of the window, and the thickening of the foot-plate. The evolutive stage of the lesions can be recognized and difficulties of the surgical approach to the fenestra ovalis can be predicted.—*H. P. Lévesque, M.D.*

KURLANDER, GERALD J., LAVY, NORMAN W., and CAMPBELL, JOHN A. Roentgen differentiation of the oculodentodigital syndrome and the Hallermann-Streiff syndrome in infancy. *Radiology*, Jan., 1966, *86*, 77–85. (From: Departments of Radiology and Medicine [Medical Genetics Program], Indiana University School of Medicine, Indianapolis, Ind.)

There are more than 100 rare head and neck syndromes which have been recently catalogued. Because of the similarity in facial appearance of some patients with different syndromes, confusion has developed which has tended to retard progress in correctly categorizing several disease entities. This communication is designed to emphasize the role of roentgen analysis as a complement to the clinical diagnosis of these rare syndromes, features of which, particularly in infancy, may appear to be limited largely to the head. Two cases, one of oculodentodigital syndrome, the other of Hallermann-Streiff syndrome, are reported with emphasis on the differential roentgen features in infancy. They are presented in detail and well illustrated.

At least 9 cases of oculodentodigital syndrome have been reported in the literature. These patients have thin noses with hypoplasia of the alae nasi and anteversion of the nostrils. Additional features include hypotrichosis with dull, dry hair, cutaneous syndactyly, and camptodactyly of the fourth and fifth fingers, with anomalies of the middle phalanges of the fingers and toes. Dental anomalies, particularly hypoplasia of enamel with exposed dentin, are frequently seen. Mental retardation and dwarfism are not typical features. The roentgenographic findings of the skull include small orbits, often with hypotelorism, and an enlarged mandible. The other bones show generalized disturbance in modeling. There is often cutaneous syndactyly of the fourth and fifth fingers, but almost constant is hypoplasia of the middle phalanges. The feet may show that the middle phalanges of several toes are absent.

The Hallermann-Streiff syndrome has been described as one of the continuum of diseases which includes craniofacial dysostosis, mandibulofacial dysostosis and cleidocranial dysostosis. The face may be small with a long, tapering, beak-like nose and an acute nasofrontal angle. The lower jaw is receding. Hypotrichosis of the scalp, atrophy of the skin of the nose, microphthalmia and bilateral congenital cataracts are encountered in almost all patients. The cataracts are unique in that they may rupture spontaneously and resorb. Additional features that may be present include blue sclerae, dental anomalies and dwarfism. The most distinctive roentgen features are of the mandible. There is hypoplasia of the rami, anterior displacement of the temporomandibular joints, and the condyles may be absent. The skull configuration is that of brachycephaly but there is delayed closure of the frontanelles and wide sutures. There is frontal and parietal bossing, the bones of the vault are thin, and the face and orbits are small. The long bones have a gracile appearance.

It can be seen that the oculodentodigital syndrome is closely related to the Hallermann-Streiff syndrome only at first glance, for critical evaluation of the clinical features and the comparison of the roentgenographic appearances expose striking differences in these two conditions. The oculodentodigital syndrome is differentiated from craniometaphyseal dysplasia and infantile cortical hyperostosis; Hallermann-Streiff syndrome from progeria.

The etiology of oculodentodigital syndrome and Hallermann-Streiff syndrome is unknown but evidence points to an autosomal recessive inheritance for the former and a dominant gene mutation for the latter. Chromosomal abnormalities have been effectively ruled out in both conditions, but teratogens and other environmental factors may play a role.—*Paul M. Kroening, M.D.*

Park, William M., and Mason, David K. Hydrostatic sialography. *Radiology*, Jan., 1966, *86*, 116–122. (From: Department of Radiodiagnosis, Western Infirmary, Glasgow, Scotland.)

Sialography is a procedure in which various diseases of the salivary glands can be diagnosed by demonstrating gross anatomic changes in their structure. The authors present in this paper a safe and quick method of sialography employing a hydrostatic technique which is suitable for routine use. A preliminary report is also given concerning an analysis of the results of studies on 108 adult patients.

The equipment used by the authors is outlined and their method of doing the procedure is described in detail. Complete filling of all ducts including the fine terminal branches is usually accomplished with the water-soluble contrast agent used. Overfilling is less likely with this hydrostatic method and patients tend to have less pain than when the hand injection technique is used. Roentgenograms are obtained in anteroposterior, lateral and lateral oblique views as soon as the passive filling phase is completed and again 5 minutes later, after the catheter has been removed from the duct. Normal glands actively expell most of the contrast material and are completely empty within half a minute. If contrast material is retained within the gland after 5 minutes the gland is abnormal.

The gland patterns in the presence of Sjogren's disease and in other obstructive conditions are illustrated.

The authors feel that this procedure, which is suitable for the routine examination of large numbers of patients, is simple to perform and free of complications.—*Donald N. Dysart, M.D.*

Rosen, Lawrence, Hanafee, William, and Nahum, Alan. Nasopharyngeal angiofibroma, and angiographic evaluation. *Radiology*, Jan., 1966, *86*, 103–107. (From: Department of Radiology, University of California Center for Health Sciences, Los Angeles, Calif.)

Two cases of nasopharyngeal angiofibroma are reported, and the value of angiography in the study of this condition is discussed by the authors. They point out that this uncommon tumor of adolescence, composed of vascular and fibrous elements, usually presents clinically with nasal obstruction and/or massive nosebleeds.

These tumors are prone to massive hemorrhage and angiograms may show a homogeneous vascular capillary blush which helps to support the clinical diagnosis without the dangers of biopsy. In addition, the blood supply to these lesions may be studied by bilateral external and internal carotid arteriography, for better control of bleeding during subsequent cryosurgery. The principal arteries supplying these neoplasms are usually the internal maxillary and ascending pharyngeal branches of the external carotid artery, and the sphenoid branches of the internal carotid artery. Bilateral ligation of the external carotid arteries prior to cryosurgery is necessary at times to reduce the operative blood loss.—*James A. Cunyus, M.D.*

NECK AND CHEST

Goff, Anne M., and Gaensler, E. A. Primary (idiopathic) pulmonary hypertension. *Med. thorac.*, 1965, *22*, 530–545. (Address: Boston University Medical Research Building, 15 Stoughton Street, Boston, Mass.)

The authors report the case of a patient with primary pulmonary hypertension in whom the diagnosis was established by open lung biopsy. The patient had a 2 year history of progressive dyspnea, and repeated cardiac catheterizations during this period revealed increased pulmonary artery pressure and normal wedge pressure. Extensive pulmonary function studies demonstrated external and alveolar hyperventilation, and excessive physiologic deadspace. Pulmonary angiograms revealed dilatation of the main pulmonary arteries with the characteristic "pruned tree" arborization pattern.

They list as criteria for the diagnosis of primary pulmonary hypertension: (1) demonstration of right ventricular hypertrophy without additional cardiac abnormality; (2) increased pulmonary artery pressure and normal wedge pressure; and (3) absence of thrombotic lesions in the pulmonary vessels at necropsy. The latter criterion can be met by lung biopsy.—*Edmund A. Franken, M.D.*

Nelson, William P., Hall, Robert J., and Garcia Efrain. Varicosities of the pulmonary veins simulating arteriovenous fistulas. *J.A.M.A.*, Jan. 3, 1966, *195*, 13–17. (Address: Major Nelson, MC, USA, Walter Reed General Hospital, Washington, D.C.)

Varicosities of the pulmonary veins are abnormally prominent and tortuous, normally draining pulmonary veins. They may be congenital in origin since they can occur in healthy young persons. They may involve any of the major pulmonary veins. Although there have been cases associated with other cardiovascular abnormalities the varicosities may be an isolated finding unrelated to pulmonary venous pressure. The abnormality is not thought to produce symptoms, nor is it accompanied by findings associated with arteriovenous shunting such as cyanosis, clubbing or bruit.

Roentgenographically, a density identified on the plain chest roentgenogram may have the appearance of a vascular shadow which tomography confirms. Fluoroscopy shows inherent pulsation with shrinkage on Valsalva's maneuver and expansion on Müller's maneuver. Pulmonary arteriography is of diagnostic value. There is filling of the large tortuous normally draining pulmonary vein during the normal venous phase of angiography. The pulmonary arteries are normal.

The natural history and hence the therapeutic approaches of this condition are as yet uncertain. Some varicosities may enlarge to the point of rupture with consequent serious or fatal pulmonary hemorrhage.

Four cases in young healthy asymptomatic men are reported.—*F. Mishkin, M.D.*

EYLER, WILLIAM R., and CLARK, MAX D. Dissecting aneurysms of the aorta: roentgen manifestations including a comparison with other types of aneurysms. *Radiology*, Dec., 1965, *85*, 1047–1057. (From: Department of Radiology, Henry Ford Hospital, Detroit, Mich.)

The development of a dissecting aneurysm of the aorta is an immediate threat to life, and successful treatment depends on early and accurate diagnosis. This is accomplished primarily by plain roentgenography and laminagraphy, following which contrast studies will demonstrate the extent of involvement as well as the physiologic alterations within the diseased and normal segments. To clarify the diagnostic and differential roentgenographic features of dissecting aneurysms of the aorta, the authors have compared the findings in 46 cases of dissecting aneurysms with 34 cases of arteriosclerotic, luetic and traumatic thoracic aneurysms.

The important roentgenographic features on plain chest roentgenograms and laminagrams include: (1) a change in the configuration of the aorta on successive chest roentgenograms, (2) aortic wall thickening shown by displacement of intimal calcification, (3) a localized hump on the aortic arch, (4) disparity in size of the descending and ascending aorta, and (5) mediastinal, lung and pleural changes.

Plain roentgenographic and laminagraphic studies of the chest allow good visualization of the aortic contour but, to be significant, any progressive enlargement of the aorta should be more rapid than expected as a result of aging. The displacement of intimal calcification away from the wall of the aorta, at least 6 mm. or more, is presumptive of a dissecting aneurysm provided that the calcification and soft tissue contour are at the same level in the body. From this standpoint lateral laminagrams are often helpful. A lack of calcification in the wall of an aneurysm also is suggestive of dissection since some calcification was present in the wall of all the other types of aneurysms here reviewed. A localized hump on the distal aortic arch was present in 22 of 39 cases of thoracic dissecting aneurysms. There was no calcification in the aneurysm wall in this group, which is in contrast to the well localized calcification in the 3 traumatic aneurysms confined to this portion of the arch. Mediastinal, lung and pleural changes are less common but include mediastinal hematoma, pleural fluid, compression or bleeding into a bronchus and widening of the paravertebral line.

The roentgenographic changes on contrast studies were documented in 27 of the 46 cases of dissecting aneurysm and include: (1) splitting of the contrast column, (2) distortion of the contrast column, (3) altered flow patterns including the failure of major vessels to fill and flow reversal or stasis, and (4) aortic valvular insufficiency.

A splitting of the contrast column, demonstrating separate channels with a wall between them, is typical and diagnostic. The false channel most commonly lies anterior and to the right in the ascending aorta, posterior and superior at the distal aortic arch and posterior and lateral to the true channel in the lower thoracic region. One of the columns of contrast material often shows a marked narrowing in caliber which can be an abrupt narrowing or a more generalized irregularity, while a defect in the intima may present as an ulcer-like projection. Nonfilling of a vital channel, especially with no symptoms of occlusion of that vessel, also is highly suggestive of dissecting aneurysm and reversal of flow in an opacified channel carries a similar implication as does renal calyceal filling without the demonstration of arterial flow to the kidney. Aortic valvular insufficiency may be the result of aortic dilatation only, but with an abrupt onset of insufficiency and disproportionate enlargement of the ascending aorta, a dissecting aneurysm must be suspected even without supporting clinical evidence.—*Edward B. Best, M.D.*

TAUBMAN, J. O., GOODMAN, D. J., and STEINER, R. E. The value of contrast studies in the investigation of aortic valve disease. *Clin. Radiol.*, Jan., 1966, *17*, 23–31. (From: Department of Radiodiagnosis, Hammersmith Hospital and Postgraduate Medical School of London, London, W. 12, England.)

The authors studied 60 patients with aortic valve disease, employing thoracic aortography and left ventricular angiocardiography. The findings were compared with those of the clinical assessment.

When clinical studies are supplemented with gradient measurements across the aortic valve, contrast studies are rarely necessary in making the diagnosis of aortic stenosis. The severity of stenosis is not reliably determined by contrast studies.

On the other hand, clinical studies may leave one in doubt as to the presence or absence of aortic incompetence, and here contrast studies will consistently clear the uncertainty, and will also give information as to the degree of incompetence.

It is essential for the surgeon to know the extent of aortic and mitral incompetence in case he contemplates surgery to the aortic valve, using cardiopulmonary by-pass. Operation on older patients with rigid, distorted and calcified valves usually will mean valve replacement.

While contrast studies are not always essential, they most often furnish useful information: (a) in younger patients; (b) when interpretation of clinical signs is uncertain; (c) when the level of obstruction is not definitely known; (d) where an associated lesion is suspected; and (e) in those patients in whom demonstration of the coronary circulation is needed. —*Samuel G. Henaerson, M.D.*

ABDOMEN

Valberg, Leslie S., McCorriston, James R., and Partington, Michael W. Bezoar: an unusual cause of protein-losing gastroenteropathy. *Canad. M. A. J.*, Feb. 19, 1966, *94*, 388–391. (From: Departments of Medicine, Surgery and Pediatrics, Queen's University, and the Special Investigation Unit, Kingston General Hospital, Kingston, Ontario, Canada.)

The authors report 2 cases of mentally retarded individuals, with a long history of ingestion of bedding, mattress stuffing, clothing, etc.

In addition to the abdominal mass noted in both patients, they were found to have severe generalized edema, and laboratory investigations revealed marked hypoalbuminemia. I^{131}-labelled human serum albumin study disclosed marked protein loss into the gastrointestinal tract apparently due to the bezoars. These were demonstrated roentgenographically.

Following removal of the bezoar, the protein loss was corrected.—*George L. Sackett, Jr., M.D.*

Moretti, S., and Fontana, G. Sull'utilità dello studio radiologico dell'ansa afferente con blocco meccanico dell'ansa efferente. (On the value of the radiologic study of the afferent loop with mechanical block of the efferent loop.) *Radiol. med.*, Nov., 1965, *51*, 1179–1189. (Address: Dott. Saverio Moretti, Via Cervese, 364—Cesena [Forli], Italy.)

Thirty-one patients with gastrectomy of the Billroth II type with antecolic end-to-side right to left gastro-jejunostomy were studied roentgenologically with a new procedure which permits a demonstration of the afferent loop. The surgical interventions were performed from 8 months to 23 years previously.

In this type of gastrectomy the afferent loop is a long blind loop excluded from the alimentary canal and is very seldom satisfactorily opacified in its entirety by the barium sulfate.

In order to overcome this difficulty the authors have adopted in 30 patients, and apparently with success, the following procedure: A two-way tube of the Miller-Abbott type is introduced into the stomach and under fluoroscopic observation is passed into the efferent loop for a length of about 8 cm. The balloon is inflated and a complete blockage of the efferent loop is obtained. About 15 cc. of a 2 per cent procaine solution is then injected through the other opening of the tube in order to eliminate any possible appearance of a spasm in the area of anastomosis between the stomach and the afferent loop. A certain amount of micropaque which varies in accordance with the size of the gastric remnant and the degree of opacification of the afferent loop, is finally injected through the tube.

In a few cases an intravenous cholangiography was performed during the procedure to evaluate possible changes in the descending segment of the duodenum, in the common bile duct and/or in the papilla of Vater.

In the opinion of the authors the procedure permits a satisfactory study of the afferent loop and the demonstration of early or late sequelae following the surgical intervention and involving either the gastric remnant and/or the afferent loop.—*A. F. Govoni, M.D.*

Dixon, John A., and Nichols, Russell L. Roentgen diagnosis of strangulation and obstruction of the intestine. *Surg., Gynec. & Obst.*, Jan., 1966, *122*, 50–54. (From: Department of Surgery, University of Utah, College of Medicine and Department of Radiology and Surgery, Dee Memorial Hospital, Ogden, Utah.)

This is a further study in the roentgen diagnosis of strangulation and obstruction of the intestine, utilizing dogs as the test animal, with emphasis on the effect of the length of the strangulated loop on changes in intestinal mobility and the effect of preoperative oral antibiotics on changes in barium transport time following strangulation.

A 2 stage surgical procedure was devised, and is described, to avoid pneumoperitoneum. The animals were placed in the following groups: no occlusion; closed loop obstruction; a short and a moderately long strangulated closed loop obstruction with preoperative antibiotics prior to strangulation; and a moderately long strangulated loop without obstruction.

Roentgenograms were taken every 2 hours and barium suspension was given at varying times following the surgical occlusion; its progress was followed every 30 minutes with cinefluorography. Postmortem correlation was made.

The findings were as follows:

1. Closed strangulated loops rarely visualized on plain roentgenograms.
2. Oral barium passed quickly to the site of the obstruction in the absence of vascular occlusion.
3. Barium was largely retained in the stomach of animals with moderately long loops of intestine with vascular occlusion of 10 hours' duration.
4. Barium was transported freely for 20–30 hours after strangulation obstruction of very short loops.
5. Prior oral broad spectrum antibiotics greatly prolonged the period of active barium transport following strangulation, suggesting tissue necrosis and bacterial action as necessary for production of changes in intestinal mobility.—*Gary M. Tolley, M.D.*

Scharer, L. L., and Burhenne, H. J. Megacolon, a nonspecific sign: clinical classification and roentgenologic differentiation. *Radiol. clin. et biol.*, 1965, *34*, 236–246. (Address: Lawrence L. Scharer, M.D., Editorial Office, Department of Medicine, University of California Medical Center, San Francisco, Calif.)

Megacolon is a descriptive term only and it refers to an abnormally dilated large bowel. There are a great number of specific conditions that may lead to megacolon.

The authors present a systemic classification of megacolon with 10 categories according to etiology. These include: (1) Obstructive ileus; (2) paralytic ileus; (3) faulty bowel habits-psychogenic; (4) endocrine abnormalities such as cretinism; (5) abnormalities of the central nervous system such as Parkinson's disease; (6) abnormalities of peripheral nervous system such as Hirschsprung's disease and reduced ganglia in Chagas' disease; (7) inflammation, as in fulminating ulcerative colitis; (8) drug administration, including ganglionic blocking agents and anticholinergic drugs; (9) miscellaneous causes such as steatorrhea; and (10) idiopathic megacolon with no definite causative factor.

The roentgenographic diagnosis is discussed and certain precautions concerning barium enemas are noted. Saline solution rather than water should be used to avoid water intoxication in some of these patients. The toxic megacolon of ulcerative colitis tends to perforate easily and these patients should not be examined by barium enema.—*Richard E. Kinzer, M.D.*

BLOOD AND LYMPH SYSTEM

Salzer, M., and Zaunbauer, W. Über Angiographie bei Extremitätentumoren. (Angiography of extremity tumors.) *Wien. klin. Wchnschr.*, Nov., 1965, *77*, 930–932. (Address: Dr. M. Salzer, Orthopädische Universitäts-klinik, Alser Strasse 4, Wien IX, Austria.)

The diagnosis of extremity tumors can prove to be difficult. Its accuracy, however, is decisive in determining the necessity of an amputation. Conventional roentgenography frequently is inadequate to rule out a malignant neoplasm of soft tissues or to differentiate between a neoplasm and an inflammatory process. Theoretically, a biopsy is sufficient to arrive at an adequate diagnosis. Its shortcomings, however, are possible spreading of tumor cells and the obtaining of misleading necrotic or other nonspecific tissues.

Angiography overcomes the disadvantages of other methods. Its technique with the Seldinger catheter or needle puncture is simple and free from hazards. Angiograms must be obtained in the arterial, capillary, and venous phase. Tumors are grouped into vascular and avascular types, either benign or malignant. Most benign tumors are avascular with the exception of hemangiomas, the rare glomus tumors, and benign osteoblastomas. Hemangiomas display varicose vessels and a rapidly appearing venous phase with an irregular staining of tissues. Semimalignant giant-cell tumors give rise to a diffuse groundglass staining already during the arterial phase.

Malignant tumors usually are highly vascular. Vessels are irregular, either stretched or tortuous, giving rise to lake formations and massive irregular tumor stain. Presence of arteriovenous fistulas causes an early venous phase. Afferent arteries are dilated. Avascular malignant tumors are uncommon and present diagnostic difficulties. Pathologic vessels are mostly in the periphery, and tumor staining is barely perceptible.

The advantages of angiography are as follows: (1) to determine the size and extent of the tumor, (2) to demonstrate a soft tissue tumor not otherwise seen, (3) only method to determine a malignant tumor with great probability, (4) to demonstrate the success of therapy, and (5) to rule out inflammatory processes and granulation tissues.

The following limitations are cited: (1) certain forms of chronic inflammation cannot be differentiated from tumors, (2) determination of smaller intraosseous lesions is limited because of the small size of nutrient vessels, (3) no specific diagnosis of soft tissue tumors is possible, (4) primary and metastatic tumors cannot be differentiated, and (5) avascular malignant tumors cannot be excluded with certainty. In view of these limitations, a biopsy is not superfluous but remains important prior to radical surgical procedures.—*Ernest Kraft, M.D.*

Tähti, Esko. Diagnostische Bedeutung der Beckenarteriographie bei Tubenschwangerschaft. (Diagnostic significance of pelvic arteriography in tubal pregnancy.) *Röntgen Blätter*, Nov., 1965, *18*, 534–536. (Address: Rö.-Abtlg. des Hebammeninstituts, Helsinki/Finland.)

The diagnosis of early tubal pregnancy is important in order to prevent serious complications. Unfortunately, however, a clinical recognition is very difficult if not impossible. Pelvic arteriography, on the other hand, is helpful as it demonstrates contrast staining of the placenta. It only fails when the placenta is detached and separated.

The Seldinger technique is applied with the catheter extending from the femoral artery upward to the bifurcation of the abdominal aorta. Twenty ml. of 60 per cent renografin is rapidly injected, while both femoral arteries are temporarily compressed to prevent an escape into the lower extremities and to secure a higher concentration in the placenta.

Fifty-five consecutive cases were examined by this method. In 34, the angiographic findings were negative for tubal implantation. Two patients actually were pregnant, but the ovum was imbedded in the distal part of the tube and had already separated at an early stage. In the remaining 21 cases, a tubal pregnancy was concluded. The diagnosis, however, was only confirmed in 17; in 3 it was false positive and in the remaining case the placenta was imbedded in a lateral horn of a uterus bicornis.

In spite of an occasional error of diagnosis, the method is valuable in recognizing tubal pregnancy at an early stage.—*Ernest Kraft, M.D.*

General

Blatz, Hanson, Heller, M. B., Kamholz, S. M., Lynch, D. E., Paul, I. R., Reich, B. S., and Smolens, M. The radiation control program. *Pub. Health Rep.*, Jan., 1966, *81*, 57–63. (Address: Mr. Blatz, Director, New York City Office, Radiation Control, New York, N. Y.)

The legal authority for the radiation control program of the New York City Department of Health is a health code. Since 1958 it has required registration of radiation installations and compliance with requirements of the National Committee on Radiation Protection and Measurements. Since 1962 the code and the agreement between New York State and the Atomic Energy Commission have provided for the regulation of radioisotopes.

The program is administered by the office of radiation control of the New York City Department of Health, which inspects the equipment and safety practices of non-industrial users of radioisotopes and about 17,000 x-ray machines in the city, processes licenses for the use of radioisotopes, responds to calls for help in radiation accidents, develops instruments and techniques, air-monitors the environment, and with a Public Health Service grant provides the instruction courses for many radiation-control employees in addition to those restricted to New York City.

The Mayor's Technical Advisory Committee on Radiation provides expert advice, and is composed of distinguished scientists in radiation and atomic energy, and practicing medical and dental radiologists. A Subcommittee reviews license applications for human uses of radioisotopes. The Mayor's Committee, the office of radiation control, and the Interagency Council on Radiation were organized in 1958 and 1959. The Council consists of the heads of appropriate city agencies who coordinate such matters as monitoring the radiologic levels of water and transporting radioactive materials.—*C. Peter Truog, M.D.*

RADIATION THERAPY

Bryce, Douglas P. Preoperative irradiation in the treatment of carcinoma of the hypopharynx. *Canad. M. A. J.*, Nov. 27, 1965, *93*, 1147–1151. (Address: 320 Medical Arts Building, 170 St. George Street, Toronto 5, Ontario, Canada.)

The results of treatment of 230 cases of carcinoma of the hypopharynx are presented. The International Classification, recently suggested by The American Joint Committee on Classification of Tumors and End Result Reporting, which is anatomic, has been used in this study.

Sixty per cent of patients present with dysphagia. This is 3 times as common as 3 other principal complaints: lump in neck, sore throat, and hoarseness. Pain referred to the ear is another important characteristic. Two-thirds of patients have symptoms 3 months before a diagnosis is made. This is unfortunate in view of the fact that 80 per cent of hypopharyngeal tumors can be detected by a simple mirror examination. Survival rates are almost doubled when the lesion is in an early stage.

Of the hypopharyngeal tumors, 60 per cent occur in the pyriform sinus. Another 24 per cent are in the post- or retrocricoid area, and 16 per cent occur in the posterior lateral wall.

Nearly two-thirds of these tumors have extended far beyond their primary site when first seen. Thirty per cent have movable cervical lymph nodes and 20 per cent show fixed lymph nodes on first examination. Five year figures show no survivals with fixed lymph nodes and 6 per cent with movable lymph nodes. However, 70 per cent of pyriform sinus patients develop cervical metastasis at some course during their disease explaining, in part, the relatively poor prognosis for these lesions under present treatment methods. Less than 3 per cent have distant metastasis on initial examination and another 10 per cent develop them during the course of their illness.

Forty-four selected patients had surgery alone. The 5 year survival was 23 per cent. Another 149 patients not considered suitable for surgery underwent radiotherapy alone, with the 5 year survival rate falling to 8 per cent.

The prognosis for pyriform sinus cancer is poor. The over-all 5 year survival rate is about 9 per cent, as opposed to 20 per cent for tumors in the other 2 regions.

Ideal therapy would eradicate the carcinoma without sacrificing normal structures. If the pharynx must be sacrificed in the treatment of hypopharyngeal cancer the patient's rehabilitation is a long and difficult process. This is unlike cancer of the larynx, where rapid recovery may occur when the larynx is removed.

Some pyriform sinus lesions can be surgically eradicated without removing the pharynx. Surgery of retrocricoid tumors almost always necessitates sacrifice of the pharynx.

If neither irradiation nor surgery used separately can bring an improvement in survival rates, it would be a logical consideration to use them in combination. Elective preoperative irradiation decided upon on the first examination and carried out with resolution has been applied in 12 cases. A full cancerocidal dose of 5,500 r, is followed in 6 weeks by complete surgical excision of the lesion and the affected structures about it. Recent work suggests that a much smaller radiation dose might be employed and surgery undertaken at an earlier date. Surgical excision is easier, if the tumor is still identifiable at operation.—*Richard Pfister, M.D.*

Madigan, J. P., and Hanson, T. A. S. Nodular sclerosing Hodgkin's disease. *J. Coll. Radiol. Australasia*, Oct., 1965, *9*, 257–262. (From: Peter MacCallum Clinic, Melbourne, Australia.)

The authors relate their experience with a recently discovered pathologic variant of Hodgkin's disease. The original work on the subject was done by Lukes in 1963. He coined the term "nodular sclerosis" to discribe the entity. The present authors report on 37 such cases out of a total of 251 cases of Hodgkin's disease, covering the period 1952 to 1962. The characteristic microscopic appearance is that of a lymph node almost totally replaced by interconnecting bands of sclerosing connective tissue, which surround small residual areas of lymphoid tissue.

Nodular sclerosing Hodgkin's disease was found to occur in a younger age group (median age 21 years) than granuloma, paragranuloma or sarcoma, the other members of the pathologic classification. It was the second most common microscopic variant found. It appeared to have a higher incidence in females than the others. Although involvement of the mediastinal lymph nodes was usually present at the time of the diagnosis, the progression of the disease to lymph nodes below the diaphragm was generally very slow. The median survival was 11 years and this compared favorably with that obtained in the paragranuloma group.

The majority of the cases were treated with 250 kv. or 4 mev. roentgen therapy delivered to the involved sites to a tumor dose of 1,500 to 3,500 rads in 3 weeks, in 12 to 15 fractions. The initial resolution of the lymph node masses seemed slower than usual, and not infrequently small inactive lymph node enlargements remained. Local recurrence, often after 5 years or more, occurred in cases given less than 3,000 rads tumor dose.

The authors believe that the different histologic pictures of the various forms of Hodgkin's disease are due to differences in host-defense mechanisms and immunologic patterns among various individuals. They suggest that this should be investigated more thoroughly.—*Kenneth M. Nowicki, M.D.*

De Giuli, Giulio. Modificazioni anatomiche e funzionali dell'intestino in corso di trattamenti radioterapici. (Anatomic and functional modifications of the intestine occurring during radiotherapeutic treatment.) *Min. radiol., fisiot., radiobiol.*, June, 1965, *10*, 231–234. (From: Istituto di Radiologia dell'Università, Firenze, Italy.)

The author discusses the anatomic and functional alterations of the intestine which he and his co-workers have observed during the past 6 years in connection with irradiation of the abdomen.

The study is divided into 6 parts: (1) clinical symptomatology; (2) coprology; (3) roentgen examination; (4) intestinal absorption and permeability; (5) dosage of pancreatic enzymes and bile acids; and (6) histology of the mucosa.

The clinical picture is characterized by an initial phase during which nausea and vomiting are frequent, followed by a phase reaching its climax towards the 3rd–4th week, when diarrhea, pyrosis and anorexia are predominant. Depending on the size of the irradiated field, an increased transit time is often noted which may affect one or more intestinal segments and may be associated with a slowing down of the emptying of the stomach. In such instances, dystonic changes may be demonstrated roentgenologically in the small intestine.

The absorption of the substances investigated (*i.e.*, oleic acid, triolein, glucose, xylose, iron, vitamin B_{12}), on the whole, appeared compromised, reaching a peak after a local dose of 2,500–3,000 r. With further increase of dose usually no further deterioration was seen; in fact, sometimes the absorption displayed a trend to return to the original value. In general, with the exception of vitamin B_{12}, no relation has been demonstrated between the site of irradiation and the site of elective absorption of the various substances.

The pancreatic enzymes may behave differently, showing, for example, an increase in diastase and a decrease in lipase. No alteration has been observed in the passive permeability of intestine investigated with I^{131} polyvinylpyrolidone.

The histologic changes were similar to those experimentally found in animals: they have also been noted in the intestinal loops outside the primary beam. In these loops, the alterations appeared to be too pronounced for the small dose of stray radiation reaching them. Some enzymatic activities persisted in the enterocytes also when marked morphologic alterations were present.

Thus, damage to the mucosa did not seem to be caused solely by the direct action of the radiation.

The clinical behavior, the data of absorption and the histologic changes pointed to a critical phase towards the end of the third week (2,500–3,000 r); after that the alterations did not become more marked; on the contrary, they sometimes became less marked.

The fact that the disturbance of absorption is usually found also when irradiation affects intestinal segments not subject to this absorption, may be due to the morphologic alterations which are observed in segments of the small intestine directly in the primary beam. The increased transit time with the accelerated motility may vary the contact between the intestinal contents and the surface of the bowel mucosa and consequently the rate of absorption, resulting in the clinical picture of inconstant digestive symptoms coupled with malabsorption.—*T. Leucutia, M.D.*

SNYDER, RUTH E. Mammography and lobular carcinoma *in situ*. *Surg., Gynec. & Obst.*, Feb., 1966, *122*, 255–260. (From: Department of Diagnostic Radiology, Memorial Hospital, New York, N. Y.)

Lobular mammary carcinoma *in situ*, as classified by Foote and Stewart, is the noninfiltrating lobular variety of breast cancer and is the type usually meant when carcinoma *in situ* is considered. The treatment at Memorial Hospital is a modified radical mastectomy. The author states that if it is managed in this way, patients are cured. If only local excision is carried out, residual disease often remains and infiltrating carcinoma may develop although perhaps not for years.

There are no regularly observed clinical manifestations of lobular carcinoma *in situ*, nor need there be any abnormality whatsoever. Before the advent of mammography, the diagnosis of carcinoma *in situ* was unsuspected preoperatively and made only after local excision was carried out for supposedly benign conditions or after the disease was found, by chance, in other areas of a breast removed because of cancer.

The author states that mammography was not used as a routine preoperative procedure on all breasts and, hence, roentgenograms have not been made of all breasts proved to contain lobular carcinoma *in situ*. She found 35 cases of carcinoma *in situ* in 35 breasts of 27 patients having mammographic coverage during a 3½ year period. It is this series that forms the basis of the present study. In 8 instances, biopsy was performed solely on the basis of the mammographic report, and in 2 additional instances, the report indicated disease in an area in which the biopsy would otherwise not have been considered. Ten or almost one-third of these lesions were detected by the roentgenologist.

The age of the patients ranged from 40 to 60 years. In most of them fibrocystic changes or indefinite thickening was noted in one or both breasts which caused them to seek the opinion of the examining physician. Because of his own equivocal clinical findings, the physician, in turn, referred the patients for mammographic examination.

Roentgenographically, in all of these breasts there was a great deal of glandular tissue of a dense, often linear character containing little fatty elements. This pattern is to be anticipated since lobular carcinoma does not occur after atrophy of the lobules. The upper outer quadrant or the 12 o'clock axis of the breast was the site of the disease in practically all instances; in 3 the disease was widespread. Although fibrocystic changes were common, this neoplasm often occurred at some distance from cysts or was found to be in localized areas of density in which markings were closely grouped.

The finding considered highly indicative of carcinoma was calcification in the form of minute punctate or linear flecks, 3 or more in number, and arranged randomly in a group, sometimes fading in distinctness at the periphery. In 9 breasts, these faint densities were formed in a definite cluster, while in 9 others a somewhat similar, though less characteristic arrangement was encountered.

Differentiation from benign diseases may be difficult, although the calcifications seen in benign entities usually seem denser and more rounded.

The author concludes that although carcinoma *in situ* may cause no recognizable abnormality in the breast, mammograms were a definite aid to diagnosis in over half of the 35 breasts of 27 patients in this study. The most important finding on the roentgenogram was a localized abnormality associated with minute calcific densities grouped in an irregular manner. Close co-operation between the surgeon, radiologist, and pathologist is necessary during all steps in the management of this disease.—*C. Peter Truog, M.D.*

RADIOISOTOPES

BULL, JAMES. Localization of cerebral lesions by radioactive isotopes. *Proc. Roy. Soc. Med.*, Dec., 1965, *58*, 1051–1053. (From: Institute of Neurology, National Hospital, London, England.)

This article summarizes the author's experience using mercury 197 or mercury 203 chlormerodrin for brain scanning in more than 700 examinations. The author indicates that the use of mercury 203 should be very much restricted because of the significant radiation dosage to the kidneys of the patient.

The 3 commonest types of brain tumors which nearly always are detected by scanning techniques include malignant astrocytoma, meningioma, and metastases. Factors which determine the detectability of these lesions relate to their size, vascularity and, in some instances, to location. Those lesions located at the base of the brain in the area of air sinuses, mastoid cells and muscle may be missed because of the high background radioactivity.

The author also mentions the use of uptake and clearance measurements of radioactive materials as a means for differentiating one type of brain tumor from another.—*Merle K. Loken, Ph.D., M.D.*

GORDON, DOUGLAS. The limitations and uses of ultrasound in localizing cerebral lesions. *Proc. Roy. Soc. Med.*, Dec., 1965, *58*, 1053–1058. (From: West End Hospital for Neurology and Neurosurgery, London, England.)

The first part of this article deals with the developmental aspects in the use of ultrasound for localizing cerebral lesions.

A summary is then given of the author's experience in 74 cases with sufficient follow-up for evaluation.

An echogram was determined to be abnormal when there was an indication of more than a 3 mm. shift of midline structures of the brain. The results obtained showed good correlation with other data including demonstration of cranial pathology at the time of surgery. This technique of echoencephalography referred to as a "A scan" which involves placement of the transducer on the lateral aspect of the head, just above the ear, does not permit data to be obtained relating to the location of any particular neoplasm.

Thus, consideration is being given to the "B scan" in which the transducer is moved in a predetermined pattern across the head. Data collected in this manner permit a two dimensional presentation to be made for localization of intracranial neoplasms.—*Merle K. Loken, Ph.D., M.D.*

MALLARD, J. R. Fundamental aspects of radioisotope scanning. *Proc. Roy. Soc. Med.*, Dec., 1965, *58*, 1058–1062. (From: Department of Physics, St. Thomas's Hospital Medical School, London, England.)

The author deals with technical considerations of detectors and display systems used in radioisotope scintiscanning. Reference is made to the use of a brain phantom in order to better evaluate the limitations of this procedure. Problems of quantitation of data by display systems with particular relation to the photoscan are discussed.

Some mention is made of the fact that radioactive materials with gamma energies in the range of 100 to 200 kev. are the best for use with most detector and collimating systems available at present. However, further study is needed to obtain the "ideal localizing materials" which give the highest concentration ratio of radioactivity in the tumor as compared to the normal brain.

Mention is also made of the newer systems for scintiphotography including the scintillation camera, the autofluoroscope and the image intensifier display system. The author indicates that systems of this type may well provide better resolution in the visualization of body organs than is possible by scanning and also that these systems are useful in the performance of various dynamic function studies.—*Merle K. Loken, Ph.D., M.D.*

LOPEZ-MAJANO, VINCENT, WAGNER, HENRY N., JR., TOW, DONALD E., and CHERNICK, VICTOR. Radioisotope scanning of the lungs in pulmonary tuberculosis. *J.A.M.A.*, Dec. 6, 1965, *194*, 1053–1058. (Address: Dr. Lopez-Majano, 3900 Loch Raven Boulevard, Baltimore, Md.)

The authors report their experience with pulmonary scans following the intravenous administration of 300 μc macroaggregated iodinated I^{131} serum albumin in 67 patients hospitalized with pulmonary tuberculosis to evaluate the extent of obliterative endarteritis secondary to chronic tuberculosis. This method has made it possible to study a large group of patients with tuberculosis with ease and little discomfort. Of the 67 patients studied, 40 had advanced disease, 19 moderately advanced disease, and 8 minimal tuberculosis. All had proven bacteriologic examination and were further classified as "active" or "inactive." Complicating lung disease was present in 31 patients. Ten patients underwent surgery and 3 of these were scanned before and after surgery, while the remaining 7 were scanned only after surgery.

The results of the examination were as follows:

(1) No complications were reported from the injections.
(2) All patients had decreased blood flow to the diseased area.
(3) The degree and extent of decreased blood flow could not be predicted from the chest roentgenograms.
(4) Associated pulmonary disease, other than tuberculosis, produced further decreased blood flow.
(5) Scans aided in preoperative evaluation of impaired blood flow and as a guide to the extent of resection needed.

(6) Postoperative scans showed a greater decrease in blood flow than would have been expected by the amount of tissue resected. After several months blood flow improved.

(7) Thoracotomy alone will produce a decreased blood flow to the operated side which lasts for several weeks.—*Richard T. Browne, M.D.*

Herrera, N. E., Gonzalez, R., Schwartz, R. D., Diggs, A. M., and Belsky, J. ^{75}Se methionine as a diagnostic agent in malignant lymphoma. *J. Nuclear Med.*, Nov., 1965, *6*, 792–804. (Address: Dr. Herrera, Director of Laboratories, Danbury Hospital, Danbury, Conn.)

There has been a good deal of activity recently in the field of radioisotope scanning of extracranial tumors with various ionic and molecular agents.

The authors describe a method which has enabled them to label for scanning 7 out of 10 lymphomas situated in the abdomen or about the trunk. Two groups of neck lymph nodes were labeled as well.

The tracer employed was Se^{75} l-methionine, administered in a dose of 250 μc per patient of average size. Scans were apparently performed 30 minutes and 24 hours after administration of the tracer.

A number of scans are shown as evidence of the fact that some lymphomatous tumors can indeed be localized by this promising new method.—*Frederick J. Bonte, M.D.*

Logan, J. W. Thyroid uptake tests in patients with malignant lymphomas. *J. Coll. Radiol. Australasia*, Oct., 1965, *9*, 263–265. (From: Wellington Hospital, Wellington, New Zealand.)

Twenty-four hour radioactive iodine uptake studies were made on 68 patients with malignant lymphomas, and compared with similar studies on 861 euthyroid patients. The initial study revealed a mean value of 24.8 ± 3 per cent for the lymphoma group, and 33.9 ± 0.8 per cent for the euthyroid controls. This difference was found to be significant at the 0.1 per cent level. Twenty-five patients, who received external radiation therapy to areas other than the abdomen, showed a significant decrease in their thyroid uptake levels at 24 hours. Eleven other patients who received a course of abdominal radiotherapy showed a significant increase in their uptake levels, as did 3 patients treated with nitrogen mustard.

Experimental work dealing with the theoretic relationship between thyroid function and lymphoma formation is discussed. One point made by the author is that malignant lymphomas are less likely to develop in experimental animals who were made hypothyroid, or given exogenous TSH. Secondly, it is pointed out that Japan, which is a goiter free area, has a very low incidence of lymphomas, whereas other areas with a higher incidence of goiter have a higher incidence of lymphomas. Finally, evidence is presented indicating that TSH is absorbed and inactivated by the thyroid gland, lymphatic tissue, the liver, and the spleen.

The author believes that lymphoma tissue may have similar properties. External radiation therapy to the abdomen eliminates the TSH inactivating substances in the liver, spleen, and retroperitoneal lymph nodes, and may explain the elevation of the uptake study after completion of the therapy.—*Kenneth M. Nowicki, M.D.*

Simpson, W. J., and Orange, R. P. Total body scanning with strontium-85 in the diagnosis of metastatic bone disease. *Canad. M. A. J.*, Dec. 11, 1965, *93*, 1237–1242. (From: Department of Radiology, The Princess Margaret Hospital, 500 Sherbourne Street, Toronto, Ontario, Canada.)

The authors report the usefulness of Sr^{85} total body scanning to demonstrate skeletal metastases that are not visible on roentgenograms and to show by autoradiographic findings the mechanism of Sr^{85} concentration.

Forty-six patients with suspected metastatic bone lesions were studied. Physical examinations were equivocal in most of these patients and none showed roentgenographic changes. In addition, 18 patients with roentgenographic changes were scanned to determine the accuracy of the technique and 1 patient with a localized skin cancer was scanned as a control.

Patients were given 200 μc Sr^{85} intravenously and scanned (total body) at 48 hours with repeat scans at 4 and 6 days in the areas of interest. The value of the 4 and 6 day scans lays in the decreased background counts and permits better localization. Scans made at different time intervals add diagnostic significance to areas of minor but repeated localization. Scanning time ranged from 45 to 60 minutes. No reactions or systemic toxicity were encountered.

When interpreting the scans, concentration in equivalent areas must be compared, right *vs.* left sacroiliac and also one vertebra *vs.* the one above and below it. Strontium is excreted in both urine and feces; thus radioactivity will be present in the colon as well as the urinary bladder.

Autoradiographs were obtained by injecting 200 to 500 μc Sr^{85} in terminal patients with known metastases shortly before death and then at postmortem examination taking thin slices of bone which were placed in plastic bags. These were then placed in close contact with radiographic film for 3 to 6 weeks. The films were developed, and the bone was decalcified and sectioned in routine fashion.

The results were as follows:

In Group 1, of 18 patients with roentgenographic changes, 16 had malignancy and of these 13 gave a positive and 3 a negative scan (2 with myelomatosis and 1 with breast cancer), while the remaining 2 patients had nonmalignant lesions and both gave positive scans (1 with osteoporosis and vertebral fracture and 1 with severe degenerative arthritis).

In Group II, of 46 patients with no roentgenographic changes, 34 had positive and 12 negative scans. Of the 34 patients with positive scans, 20 had proven metastases to the area of localization, while 3 had no lesion in the area of localization at 1 year. The scans of the remaining 11 patients were inconclusive as no further roentgenographic examinations were done; on the 5 patients who died no autopsy was performed. Of the 12 patients with negative scans, 3 were subsequently proven to have metastases (2 from carcinoma of breast and 1 from myelomatosis), 6 had no bone metastases at 1 year or more, while in the remaining 3 the result was inconclusive, as less than 1 year had elapsed since the scans were performed.

Autoradiographs were not successful in most cases in demonstrating the sites of Sr^{85} localization and this was felt to be due to the very diffuse skeletal involvement. Those radiographs which did demonstrate concentration did so in areas of "tumor osteogenesis," *i.e.*, in areas where tumor cells were associated with areas of new bone formation.—*Richard T. Browne, M.D.*

Mailloux, Lionel U., and Streeto, James M. The effect of prior vitamin B-12 administration on the Schilling test. *Am. J. M. Sc.*, Dec., 1965, *250*, 697–699. (From: Department of Medicine, Hartford Hospital, Hartford, Conn.)

Among the factors that may lead to the improper interpretation of the Schilling test are impaired renal function, failure to obtain a complete urine collection, and a poor testing technique. According to the authors, there has been only one previous report in the literature of urinary Co^{57}-B_{12} excretion being suppressed by parenteral B_{12} administration in the days immediately preceding a Schilling test.

This report deals with 12 patients who had a normal Schilling test as determined by standard technique and then were restudied following the intramuscular injection of nonradioactive B_{12} (1,000 μg./day) for 3 to 5 days prior to a repeat Schilling test.

The results are reported as showing that 10 of the 12 patients had reduced urinary excretion of Co^{57}-B_{12} ranging from 13 to 69 per cent of the value obtained in their first Schilling test. Two patients had increased excretion of Co^{57}-B_{12}. No explanation of the mechanism is given.—*Richard T. Browne, M.D.*

Horwitz, Norman H., Lofstrom, James E., and Forsaith, Ann L. The spintharicon: a new approach to radiation imaging. *J. Nuclear Med.*, Oct., 1965, *6*, 724–739. (From: William Beaumont Hospital, Royal Oak, Mich.)

Isotope radiography may well be in a transitional stage between moving and stationary imaging devices.

The authors point out that moving devices require a relatively long time to perform the imaging process, and make an additional valuable point in that a line scanner is utilizing only the radiation within its limited field of view at any particular time and thus does not respond to radiation originating from all parts of the volume of interest. This necessarily results in information being acquired in a serial manner, so that there is a temporal lag in data acquisition between the volumes covered at the beginning and the end of the scan.

The Anger camera, and its various modifications, and the image tube adaptation of Ter-Pogossian are the principal known stationary imaging devices, but to this small group must be added the authors' instrument, which works on a novel principle, employing a discharge tube. This tube consists of 2 electrodes in a gas-filled envelope. Free electrons are produced by the interaction of radiation with the cathode or the filling gas, and further free electrons are caused by collisions of the initially liberated electrons as they fall through an electrical field. In a properly constructed tube an avalanche of electrons will produce a spark, the light from which may be photographically recorded.

The advantages of the spark discharge tube in isotope radiography are that the spark discharge may be highly localized to the site of gamma ray interaction, and the fact that the spark is of sufficient luminosity to permit photographic localization of the interaction site, and perhaps even visual observation as well.

The authors describe in detail the construction of their spark discharge (spintharicon) chamber, as well as the construction of their honeycomb collimators. Both early and recent clinical results in the scanning of thyroid glands are illustrated and are encouraging. Those who are interested in isotope radiographic equipment should read this article in detail.—*Frederick J. Bonte, M.D.*

CHEMOTHERAPY

Ödman, Per. Methode einer perkutanen, selektiven intraarteriellen Infusion von Zytostatika. (A method for percutaneous, selective intraarterial infusion of cytostatics.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Dec., 1965, *103*, 643–652. (Address: Röntgenabteilung Norrköpings lasarett, Norrköping/Sweden.)

Various cytostatics were used in the treatment of malignant growths or metastases found in 29 patients. The drugs were administered in combination with or following irradiation of the involved sites. Infusion was preferable to perfusion. After selective arteriography determined the site and extent of the lesion, it was a simple matter to instill the desired cytostatic or combination of drugs.

The author's preference was Sendoxan, Pharmacia (cyclophosphamid), the dose being 200 mg. in 400–500 cc. of physiologic saline. Other cytostatics were: 20 mg. Tifosyl, Astra (triethylenetriphosphoramide), 50 mg. Methotrexate, Lederle (4 amino-N-10 methylfolic acid) in a 1,000 cc. saline solution, combined with 24 mg. Leucovorin, Lederle (formyl tetrahydropteroylglutamic acid) in 1,000 cc. saline solution, intravenously.

The drip infusion should be performed at a rate of at least 15–20 drops per minute to avoid the possibility of coagulation in the polyethylene catheter. As much as 3,500–4,000 mg. Sendoxan was infused over a 5–7 day period without producing any thrombocytopenia or significant leukopenia. A maintenance dose of 50 mg. 3 times daily or 400 mg. per week of Sendoxan, intravenously may be carried on for several months, provided that the leukocyte count is routinely checked. The larger doses of Sendoxan may cause a temporary, generalized loss of hair, elevation of temperature, or circulatory spasm and insufficiency. Tifosyl is not recommended because of its tendency to cause bleeding. Methotrexate may cause thrombocytopenia but has little effect on the leukocyte count.

Of the 29 patients treated, 18 were terminal cases and died shortly after onset of cytostatic therapy; 7 patients were rendered operable; in 4 patients, spread of metastases was prevented. In all, 14 patients were considered improved as indicated by regression of the primary tumor or local metastases, disappearance of malignant lesions, or reduction in ascites. In most instances, it was felt that the cytostatics enhanced the effects of the simultaneous or prior irradiation.

Some details of 3 patients were documented in this article: (1) A 45 year old woman developed axillary metastases 1½ years following mastectomy. After irradiation and Sendoxan, the lesions disappeared and she has remained "cancer-free" for 5 years. (2) A 60 year old woman had a recurrence under the abdominal scar after a total gastrectomy. She received 1,600 r to each of two anterior and posterior fields as well as 3,500 mg. of Sendoxan by infusion into the celiac artery. At subsequent surgery, no tumor cells were found and she has remained well for 4 years. (3) A 25 year old male with metastases from a melanoma remained well for 4 years after 2,800 mg. of Sendoxan infused into the internal iliac artery.—*J. Zausner, M.D.*

Silberman, Harold R., McGinn, Thomas G., and Kremer, William B. Control of fever in Hodgkin's disease by indomethacin. *J.A.M.A.*, Nov. 8, 1965, *194*, 597–600. (Address: Dr. Silberman, Duke Hospital, Durham, N. C.)

Indomethacin (Indocin), a new nonsteroid, anti-inflammatory, antipyretic agent was evaluated in 9 patients with fever due to Hodgkin's disease.

Fever, without obvious infection, has been recognized for many years as a cardinal symptom of Hodgkin's disease. It is present in 30 to 60 per cent of patients as part of the initial manifestation of this illness. It occurs as a distinctive relapsing form (Pel-Ebstein) and the more common typeless form, manifested as continuous, remittent, intermittent, or occurring in combinations. It usually means active occult disease. Some patients continue with fever, while responding to cytotoxic agents with obvious lymph node or organ regression.

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All 9 patients had significant and prolonged fever despite treatment by cytotoxic chemotherapy, salicylates and steroids. Each patient treated with indomethacin had prompt relief of fever within 24 hours. When this agent was omitted, fever promptly recurred in 12 to 24 hours, but again responded to reinstitution of therapy.

The authors stress the exclusion of complicating infection, and an adequate trial of appropriate chemotherapy before using a nonspecific antipyretic. —*Richard Pfister, M.D.*

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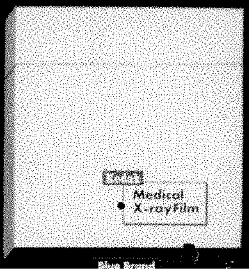

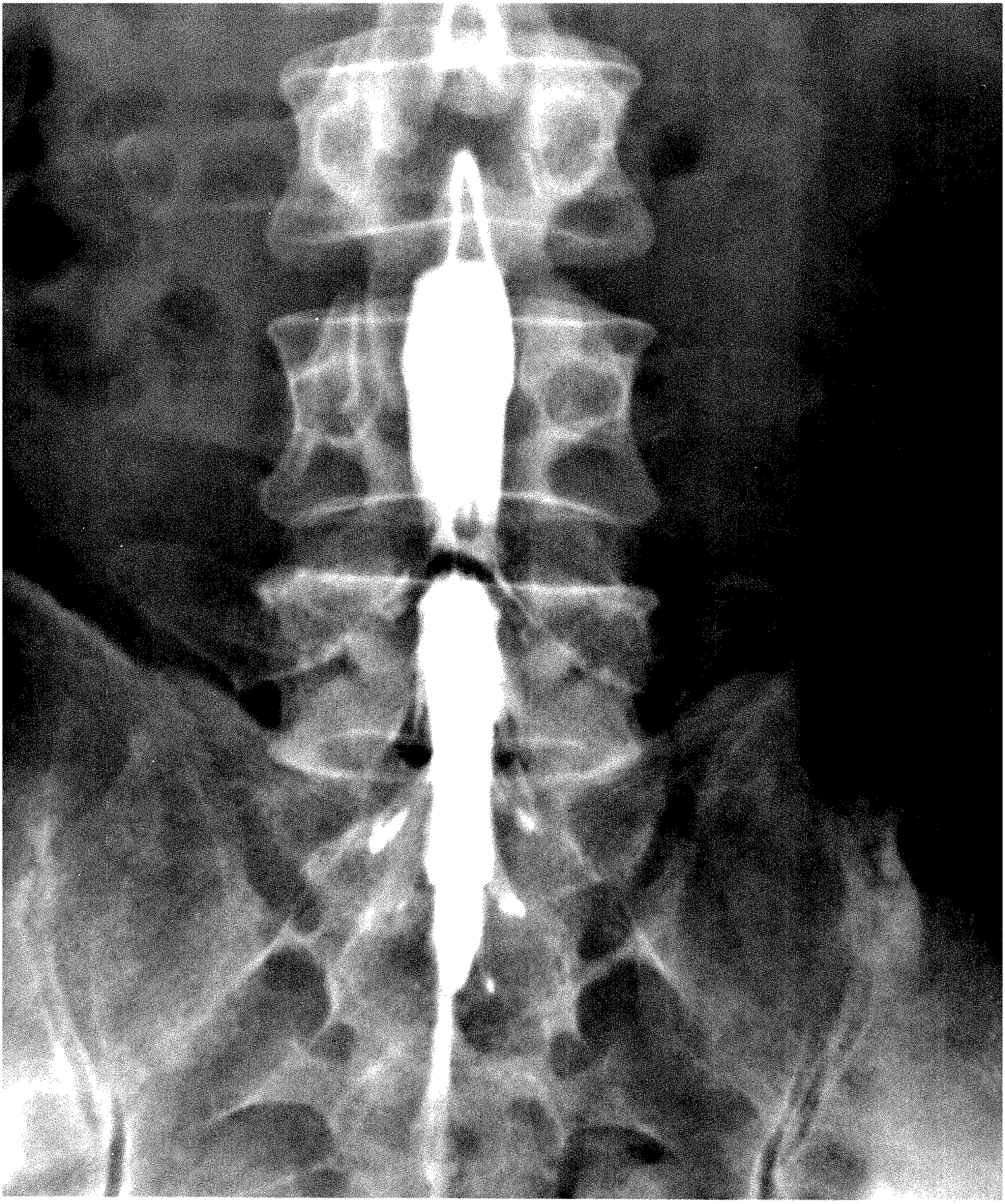

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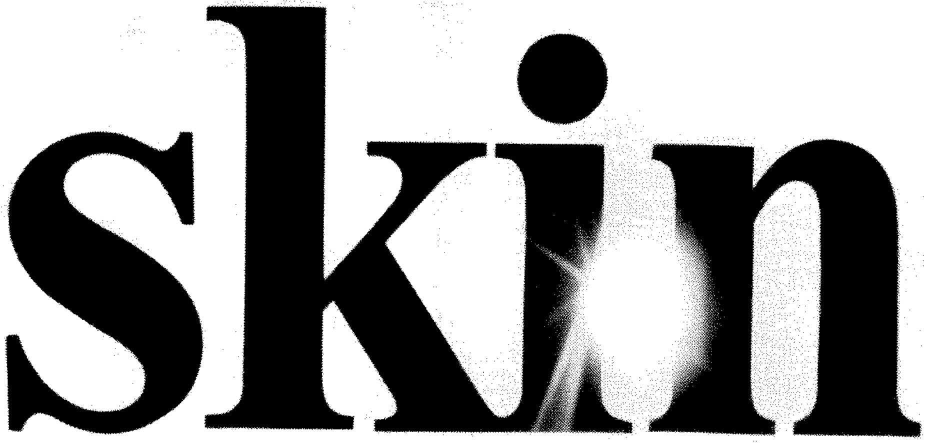

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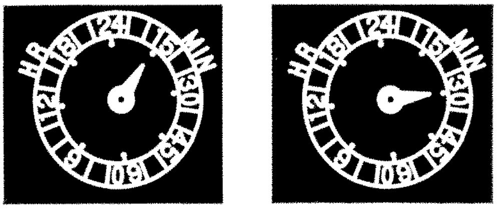

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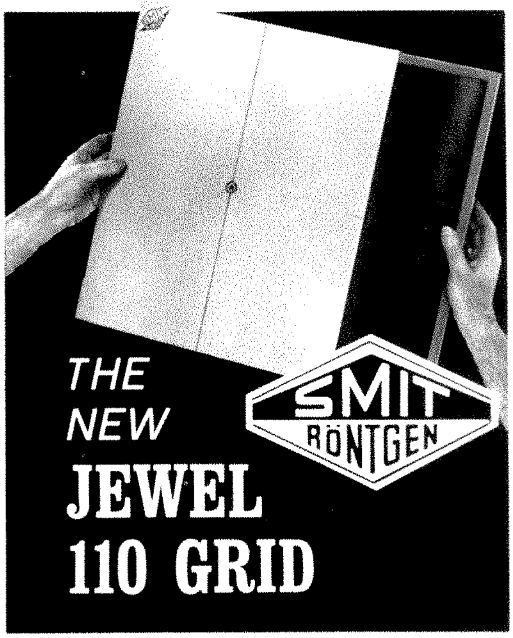

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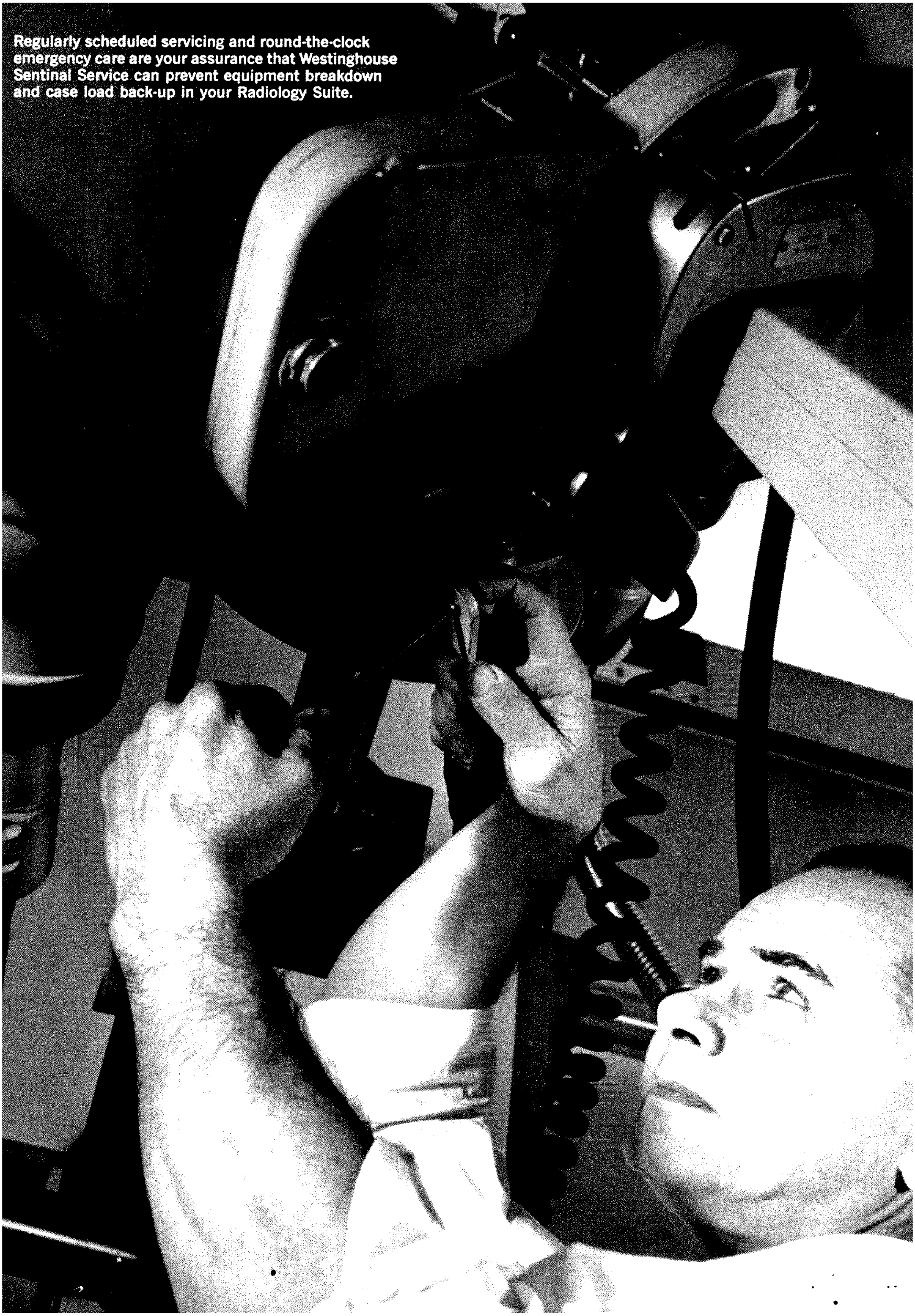

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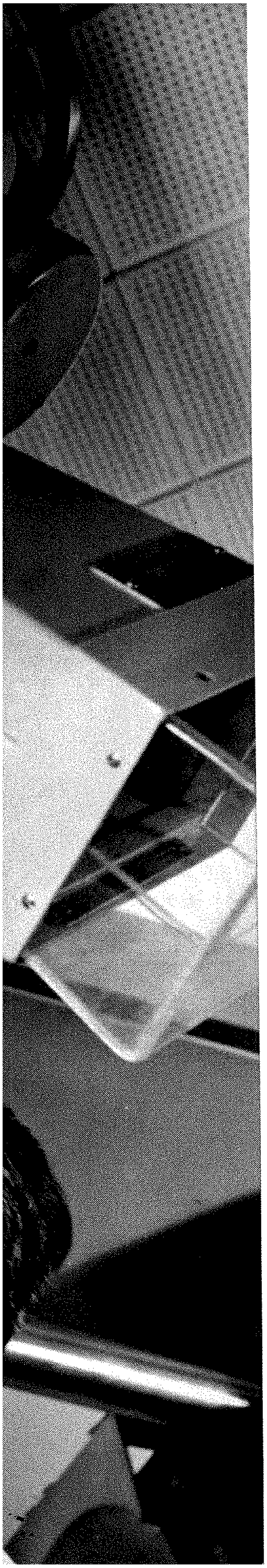

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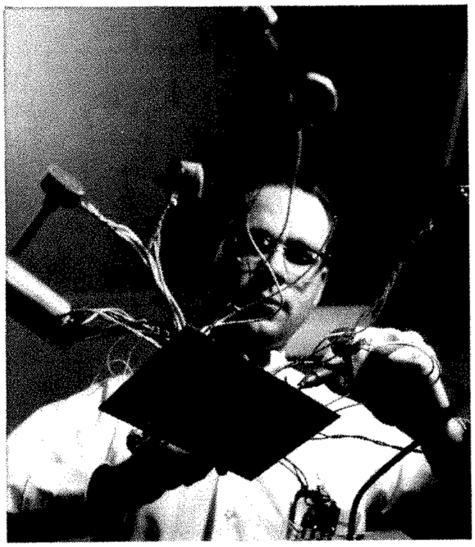

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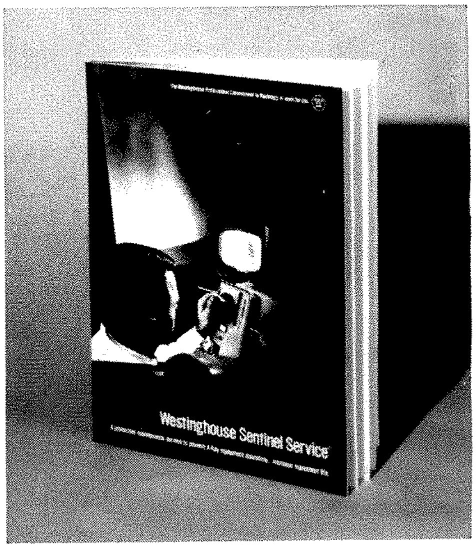

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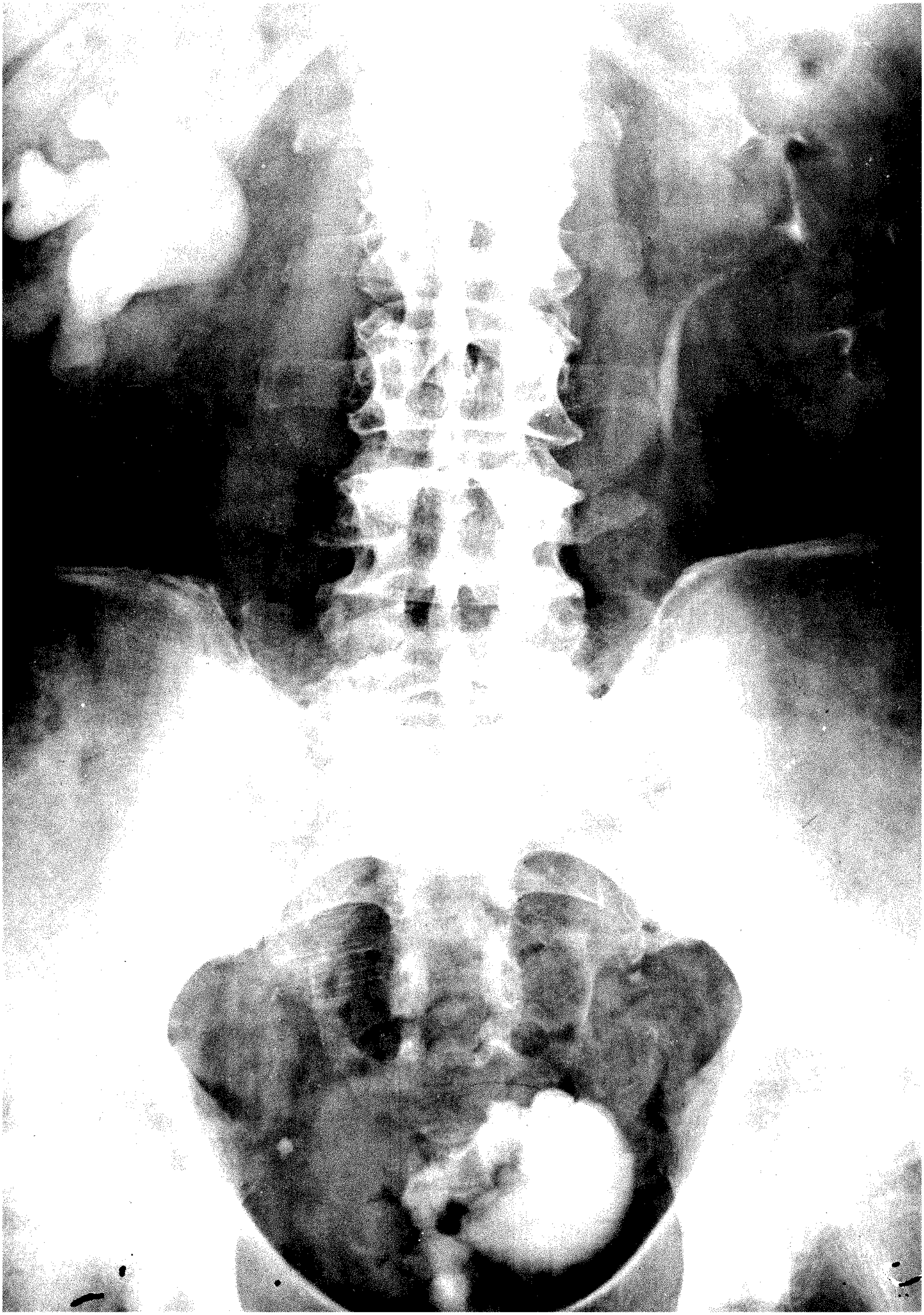

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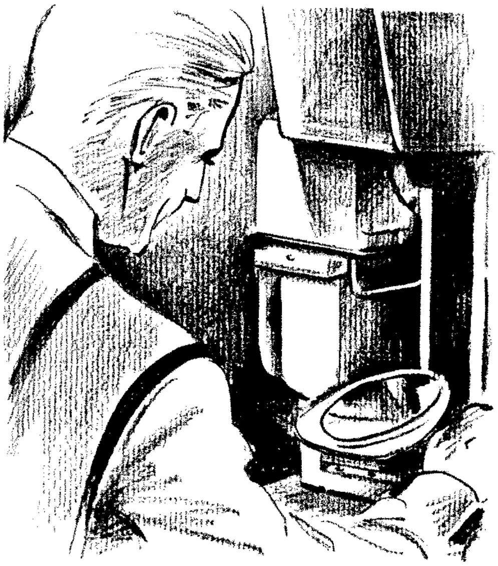

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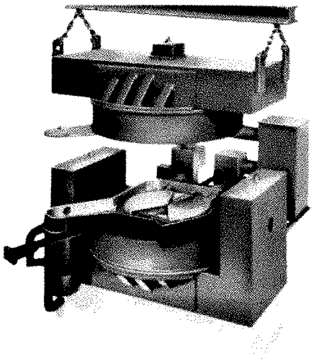

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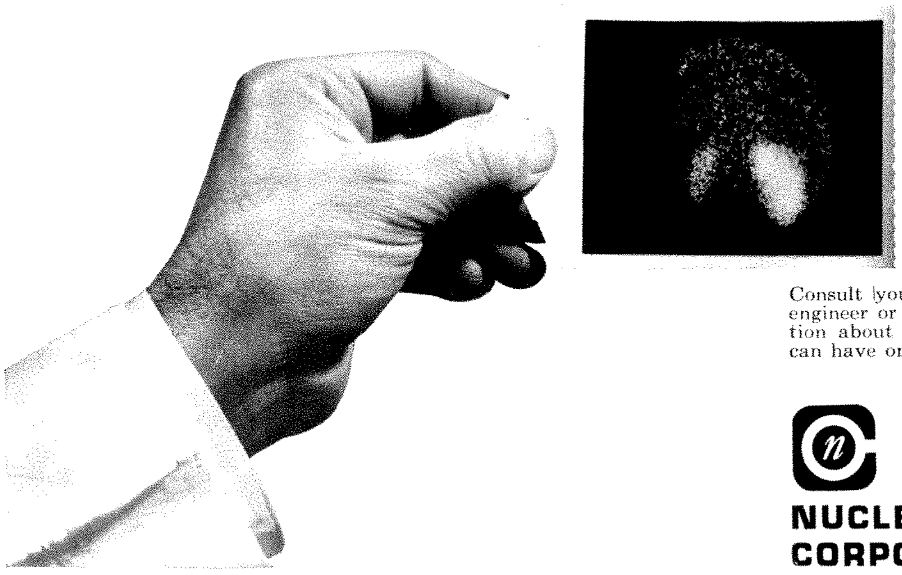

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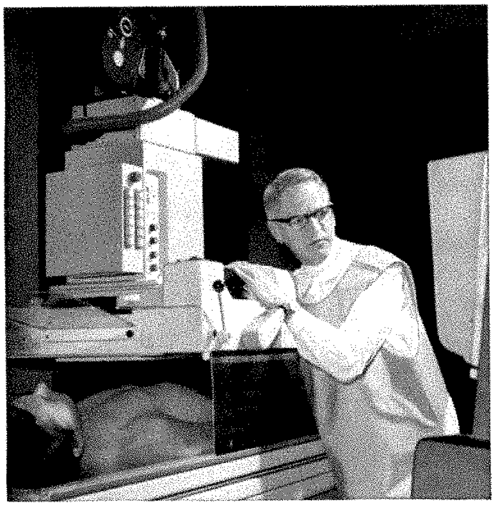

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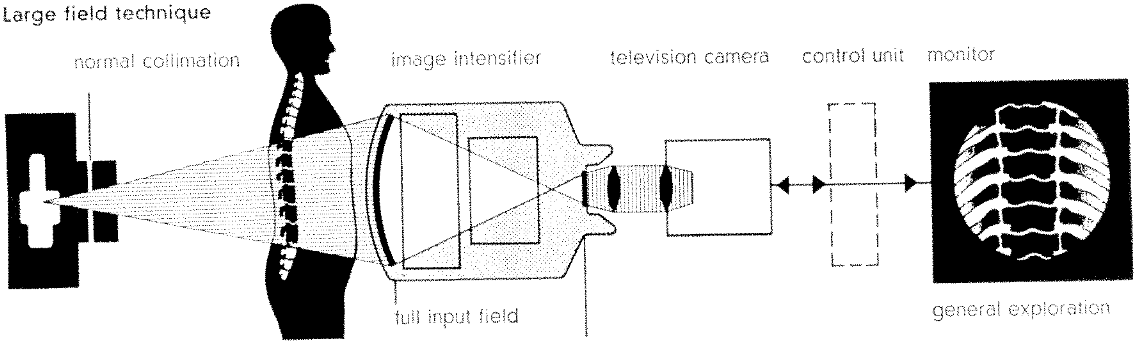

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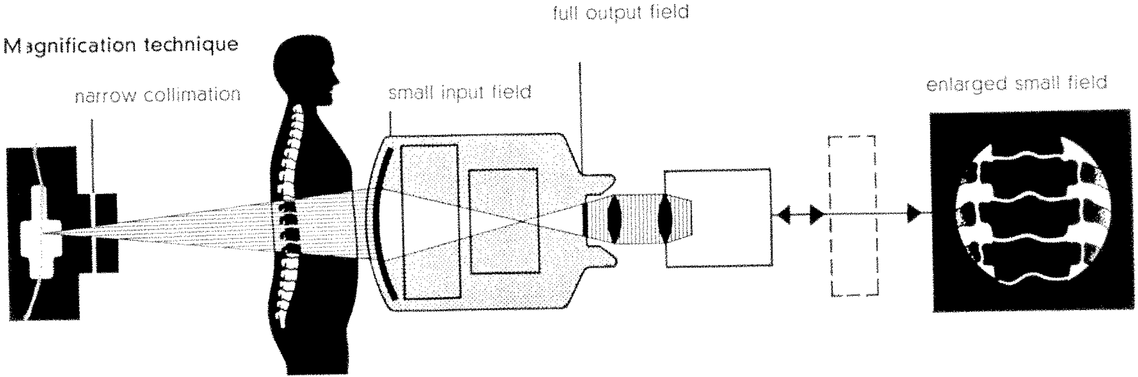

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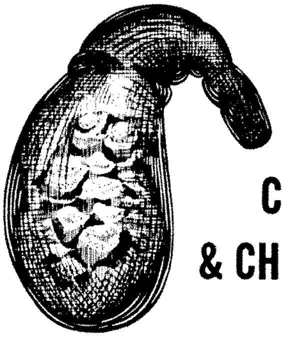

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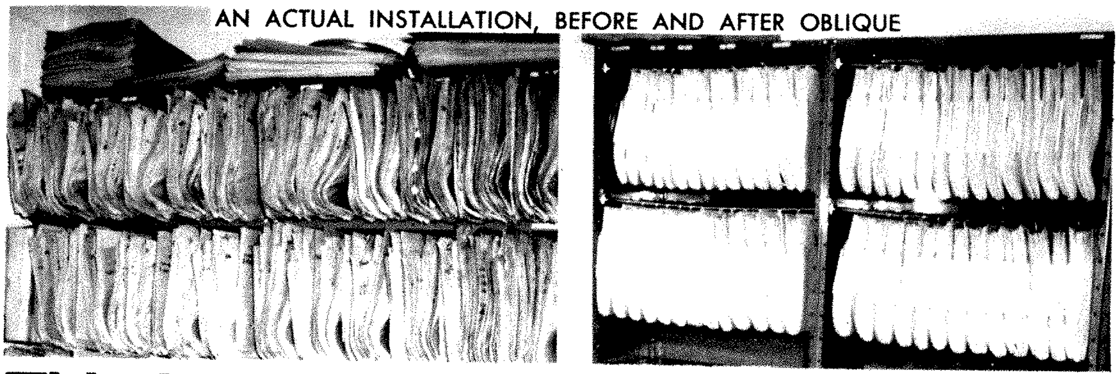

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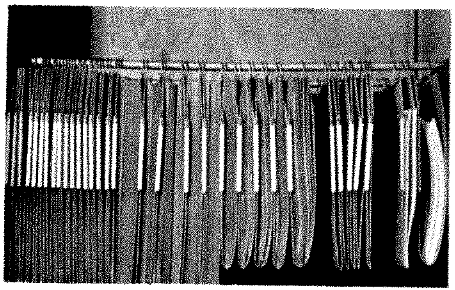

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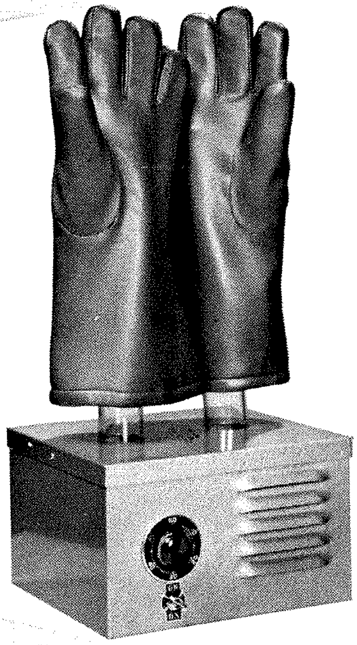

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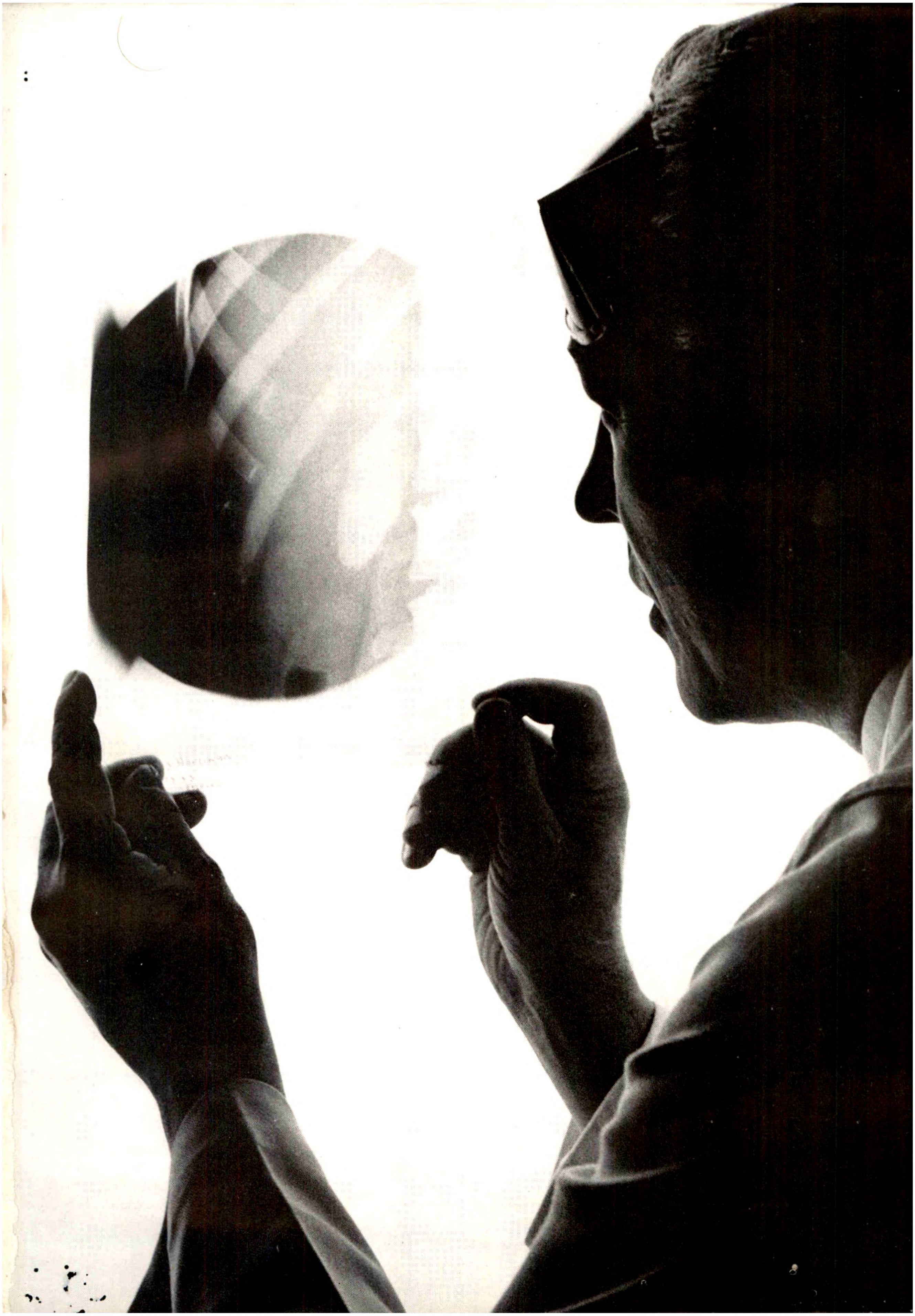

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